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Projecting the Demographic Impact of AIDS

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and
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A simple model that simulates the spread of AIDS is used to generate estimates of deaths from AIDS.

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This paper — a product of the Population Policy and Advisory Service, Population and Human Resources Department — is part of a continuing effort in the department to produce up-to-date population estimates and long-run population projections. Copies of the paper are available free from the World Bank, 1818 H Street NW, Washington DC 20433. Please contact Otilia Nadora, room S6-065, extension 31091 (July 1992, 57 pages).

A simple model that simulates the spread of AIDS is used to generate estimates of deaths from AIDS, which are incorporated into population projections covering 20 years. Preliminary results for one country are shown — not firm estimates, as the model has several arbitrarily set parameters.

The results suggest that the number of infections and deaths could be extremely large, even if transmission of the human immunodeficiency virus (HIV) is substantially reduced. In five years, deaths in a single country will be in the tens of thousands, and after 20 years could be in hundreds of thousands and still rising. Nevertheless, the impact on population size appears small. Bulatao and Bos discuss why these results should not be entirely trusted, and what work remains to be done.

Where HIV is relatively widespread, changes in sexual behavior, particularly increases in condom use, are essential to reduce the scale of the epidemic. Earlier changes are more effective than later changes. But across countries with different levels of prevalence and sexual activity, changes in sexual behavior produce similar effects.

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INITIAL INVESTIGATION OF THE DEMOGRAPHIC IMPACT OF AIDS

Rodolfo A. Bulatao

> Though it has many omissions and contains much that is apocryphal, or at least wildly inaccurate, it scores over the older, more pedestrian work in two important respects.
>
> First, it is slightly cheaper; and second, it has the words DON'T PANIC inscribed in large friendly letters on its cover.
>
> --Douglas Adams, *The Hitchhiker's Guide to the Galaxy*

This note illustrates an approach to investigating the demographic implications of the acquired immunodeficiency syndrome (AIDS) pandemic in Africa. A simple model is constructed to simulate the spread of AIDS. The model is used to generate estimates of deaths from AIDS, which are incorporated into population projections covering 20 years. Preliminary results from applying the model to one country are shown.

These results are not meant as firm estimates, based as they are on a simple model with several arbitrarily set parameters. The results do suggest that the number of infections and deaths could be extremely large, even if transmission of the human immunodeficiency virus (HIV) is substantially reduced. In five years, deaths will be in the tens of thousands, and after 20 years could be in hundreds of thousands and still rising. Nevertheless, the impact on population size appears small. These results hold only, however, under the conditions specified below.

This note describes the model, its implementation, the simulation of experience for one country, and population projections for that country. Finally, the reasons why these results should not be entirely trusted are discussed, and areas where work is needed indicated.

MODEL

Three processes in the spread of AIDS are represented in the model: the incidence of HIV infection within the population; the development of frank AIDS among those infected; and deaths among those with AIDS. For each process, adults (15 years old and over) and children are treated separately. Each process will be discussed and the linkage with population projections considered.

Adult transmission

Adult HIV infections are assumed to be due to sexual contact or to injections and transfusions combined. (Only injections will be referred to below, though parameters are set to some extent taking transfusions into account.) Four partially overlapping risk groups are distinguished: all adults, as potential injection recipients; women, assumed to be all heterosexual; heterosexual men; and homosexual men.

The number of seroconversions (O_{rt}) in a given year (t) in each of the three risk groups (r) distinguished by sex is determined following these steps. First, some constant rate of transmission of the virus (T_r) between one infected and one uninfected partner is assumed per sex act. This rate varies across but not within risk groups. Second, this rate is adjusted by taking into account the average number of unprotected sex acts per year (S_r) between new sexual partners. Unprotected sex acts are those in which condoms are not used. Third, both the infected and the

uninfected in each risk group are divided into three subgroups (s)--high, medium, and low--by frequency of obtaining new sexual partners. A single index number (F_{rs}) for new sexual partners per year is applied to all members of one subgroup. Fourth, these index numbers are used as weights in determining the proportion (R_{rt}) of pairings of new sex partners in a given year that involve an infective partner. The formula for homosexuals, for instance, is

$$R_{rt} = (\Sigma_s F_{rs} A_{rst}) / (\Sigma_s F_{rs} [A_{rst} + M_{rst}])$$

where M represents seronegatives and A the asymptomatic (seropositives who have not developed frank AIDS). Frank AIDS, though not seropositivity, is assumed to limit sexual contact. Fifth, the seroconversions in each subgroup (O_{rst}) are determined from the number of seronegatives and the probabilities and proportions estimated in the preceding steps, using the formula

$$O_{rst} = M_{rs,t-1} \{P_{t-1}\} \{1 - (1 - \Sigma_s O_{1st} / M_{1s,t-1}) (1 - R_{r,t-1} [1 - (1 - T_r)^{St}])^{Frs}\}.$$

P_t is the probability of adults surviving all other causes of death and O_{1st} and M_{1st} stand for seroconversions and seronegatives in the injection risk group.

The seroconversions due to injections are similarly estimated, though without adjusting, as in the formula above, for seroconversions due to other causes. In this case, the index number refers to number of injections per year. The frequency-of-intercourse parameter is not used; instead, another parameter is introduced for proportion of needles sterilized.

All the transmission parameters stay constant up to the present, but some are allowed to change in the future, decreasing or increasing by fixed annual percentages. The parameters that may change are the proportion of needles sterilized, the number of injections per year, the frequency of unprotected intercourse in one year in each new pairing, and the number of new partners per year.

Adult progression

Among adults infected, the cumulative probability of contracting frank AIDS by year t is assumed to follow a logistic pattern, of the form

$$H = k / [1 + \exp (a + bt)],$$

from which the year-by-year hazard is derived. The proportion of those infected who eventually develop AIDS is set by k, which lies between 0 and 1. With any reasonable parameters, this equation gives an essentially zero hazard of developing AIDS 20 years after initial infection, if not earlier. For convenience, the hazard is therefore fixed at zero from year 20 on. Other possible curves to represent the incubation period are considered in Appendix A.

The cumulative probability of adults dying from AIDS, among those with frank AIDS, is assumed to follow a similar logistic pattern. The likelihood of dying from AIDS in a given year is fixed at zero 20 years after developing AIDS and for subsequent years.

Children

Among children, HIV infection is assumed always to take place perinatally. The number of infants infected is the product of the number of infected women of reproductive age, a fertility rate applicable to women of these ages, an adjustment factor to allow for these women having fertility either lower or higher than typical, and a transmission rate. As with adults, the transmission rate is fixed up to the present but can decline by a constant annual percentage in the

future.

The proportions progressing to frank AIDS and dying from the disease are estimated by applying logistic functions similar to those for adults, with the parameters set to allow substantially faster progression among children.

Demographic projections

The model covers two periods: a simulation period of 15 years, meant to cover the initial phase of the epidemic, during which demographic rates are not affected by AIDS; and a subsequent 20-year projection period, during which they are. Cases and deaths are estimated year by year, but demographic estimates are made quinquennially.

Mortality from other causes is taken into account in both periods by applying calendar year-specific mortality rates. A single average mortality rate is used for all adults, but for children age-specific rates are used. The model assigns some probability to individuals dying simultaneously from AIDS and from other causes. These deaths would occur in any case and are not counted as AIDS deaths.

Quinquennial age-specific death rates from AIDS are estimated using the age distribution of estimated annual deaths for children and applying an age distribution of infected adults to assign adult deaths to specific age groups. The distribution of adult deaths is shifted toward older ages relative to the distribution of those infected, on the assumption that deaths will eventually occur, on average, roughly a decade after infection.

The estimated mortality rates from AIDS are combined with projected mortality rates from all other causes and entered into a linked microcomputer package, PROJPC, which is generally used for World Bank demographic projections. Iteration between the Lotus worksheets and PROJPC is necessary to complete the projections.

Many simplifying assumptions are made in this model and will be pointed out in detail in the discussion section below.

RESULTS

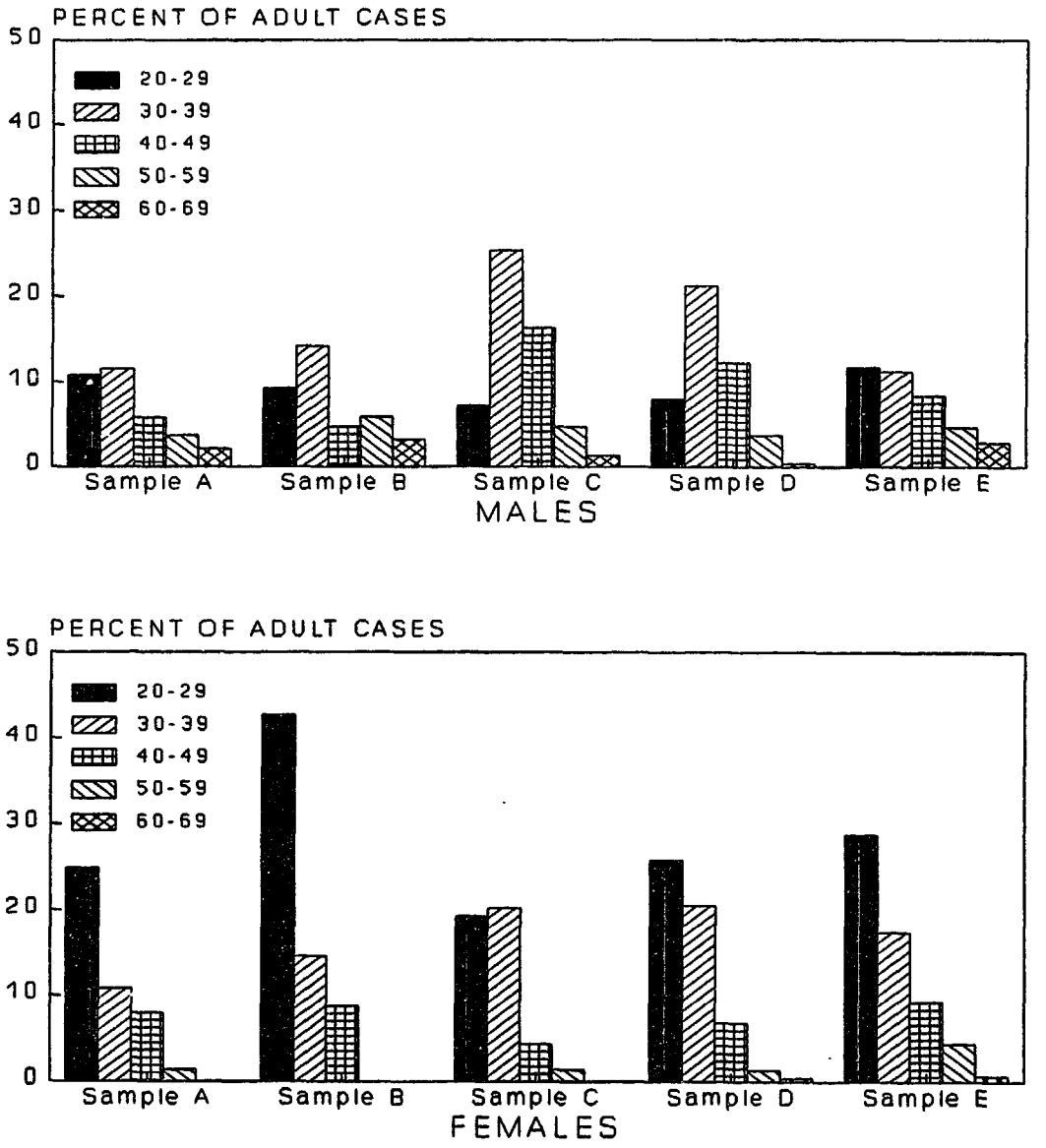
Simulation

The model was applied to the experience of Zaire from 1970 to 1985. First I explain why the place and the period were chosen; next I consider the age-sex distribution of seropositives, which is a key to several parameters; then I specify the demographic assumptions as well as the parameters for the spread of AIDS; and finally I compare model-generated estimates with reported estimates.

More data relating to AIDS exist on Kinshasa, Zaire, than on any other place in Africa, enough to allow testing of various plausible estimates of the model's parameters. The year 1970 is chosen as a starting point, though an earlier year is possible: a 1959 blood sample showed antibodies to HIV, and seroprevalence was reported at 0.2 percent for a 1970 sample of pregnant women in Kinshasa.

Figure 1 compares five age-sex distributions relating to AIDS for Kinshasa: (A) of seropositives in the general population; (B) of seropositives among hospital workers; (C) of AIDS

Figure 1 Five age-sex distributions for seropositive (but healthy) adults or adult AIDS cases: Kinshasa, Zaire



Notes and sources: Age distribution calculated from Zaire 1985 age-sex structure and:

A seroprevalence among 5,099 healthy persons, 1985-85 (Quinn et al. 1986)
B seroprevalence among 2,384 hospital workers, 1984 (Mann et al. 1986b)
C reported AIDS cases, July 1984-February 1985 (Mann et al. 1986e; Quinn et al. 1986)
D 500 reported AIDS cases, August-December 1985 (Quinn et al. 1986)
E 500 reported AIDS cases, August-December 1985, "aged" five years.

cases in 1984-85; (D) of AIDS cases later in 1985; and (E) of seropositives in the general population adjusted by being "aged" five years (Mann et al. 1986b, 1986e; Quinn et al. 1986). These are all small samples and do not refer to exactly the same phenomena; close consistency should not be expected.

Three points of difference among the samples are the proportion of females 20-29, the proportion of females 30-39, and the proportion of younger males among those affected. For females 20-29, sample B (the hospital-worker sample) is discrepant. There is no obvious explanation, and the other three samples agree among themselves. For females 30-39, the two samples of AIDS cases (C and D) give higher proportions than the samples for seroprevalence. AIDS cases might be expected to be slightly older than healthy seropositives, since AIDS takes several years to develop. To allow for this, the distribution for sample A (the only one given in five-year age groups) is repeated as sample E, but with the 15-24 cohort standing in for 20-29, 25-34 for 30-39, etc. This results in a closer match with samples C and D.

For males the discrepancies are more troubling. A similar adjustment reduces but does not remove the main discrepancy: a substantially greater proportion between the ages 30 and 49 among AIDS cases than among seropositives. This may be due to a greater probability that seriously ill males in these ages, even rural males, will be brought to a hospital, or that their illness will be diagnosed as AIDS. On the assumption that many such biases exist in counts of AIDS cases, the seroprevalence data (sample A) will be relied on here, and are given in full in Table 1.

Table 1. Assumed distribution of seropositives (percent)

Age	Male	Female
0	3.95	3.92
1-4	1.70	2.73
5-9	1.81	2.71
10-14	1.51	12.17
15-19	4.66	10.69
20-24	4.65	9.15
25-29	3.89	4.63
30-34	5.00	3.93
35-39	4.15	3.42
40-44	2.51	2.87
45-49	2.04	0.61
50-54	1.63	0.49
55-59	1.28	0.00
60-64	0.96	0.00
65-69	0.68	0.00

Source: Estimated from data on 5099 healthy persons in Kinshasa (Quinn et al. 1986), applying 1985 Zaire age-sex distribution.

Only adults are compared in Figure 1. The proportion of children in sample A--20.6 percent, as Table 1 shows--is considerably greater than that in sample D, where children were only 6.7 percent of all cases. The higher number appears preferable (Mann et al. 1986d), the lower one being at least partly explainable if children are less likely to be hospitalized and to have their illness properly diagnosed.

A fully reliable age distribution for Zaire as a whole is not available--1984 census reports do not provide this information. Demographic assumptions are taken, therefore, from Bank estimates and standard Bank projections. Between 1970 and 1985, population is assumed to grow from 19.5 to 30.6 million, with the proportion adult falling from 56 to 52 percent. The proportion urban, estimated from figures for 1955, 1973, and 1984, rises between 1970 and 1985 from 23 to 41 percent.

Mortality is defined by Coale-Demeny model North life tables, around levels 13 and 14, giving life expectancies between 48 and 52. Mortality rates from all causes except AIDS for adults affected by AIDS come from age-specific life table rates weighted by the age distribution (taking adults only) in Table 1. In this distribution, women of reproductive age are 55 percent of adult seropositives. The age distribution of these women is used to calculate a weighted fertility rate from model age-specific rates. Fertility is assumed not to change. None of the demographic parameters is assumed to have been affected by AIDS in the simulation period.

HIV infection incidence in 1970 is arbitrarily set at 0.1 percent of urban adults, below the rate for pregnant Kinshasa women earlier noted. The assumed transmission rates per sexual act with an infected partner are set at .0026 for male to female as well as for homosexual transmission and .0016 for female to male transmission. One small study of American female partners of male seropositives gives a confidence interval for male to female transmission per sexual act of .0001 to .0026 (Padian et al. 1987). The upper limit is used here on the assumption of more efficient transmission in Africa, possibly because of untreated sexually transmitted diseases.

Given the paucity of data on sexual behavior, parameters relating to it are set arbitrarily. High, medium, and low subgroups are assumed to include 10, 30, and 60 percent respectively of females and heterosexual males. For homosexuals, assumed to be 5 percent of adult males, the distribution is 20, 30, and 50 percent. Average numbers of new partners per year for high, medium, and low subgroups are set at 21, 3, and 0.2 among females; 18, 3, and 0.2 among heterosexual males; and 36, 5, and 0.3 among homosexuals. These numbers appear reasonable and, as will be shown, produce reasonable results, but many alternative combinations of parameters could of course give the same results.

For transmission through injections, parameters were also chosen arbitrarily: .003 for the transmission rate per injection; 20 percent of needles sterilized; 10, 20, and 70 percent of the risk group in high, medium, and low subgroups; and 30, 5, and 0.5 as the average annual frequency of injections in each subgroup.

The transmission rate from a woman to her baby is set at 0.70, and no fertility adjustment factor is used. A transmission rate of 0.50 is more commonly cited but may be conservative. A sample from a well-child clinic in Kinshasa implies a transmission rate of 0.69, and a parallel sample from measles and general pediatric hospital wards implies a rate of 0.80 (Mann et al. 1986b).

For progression to AIDS, the parameters are based on the analysis in Appendix A, with one modification. Following the argument that the transfusion-induced AIDS cases considered

there probably had a shorter than normal incubation period. Higher values than estimated of the parameter a in the logistic equation are used--6.0 and 4.0, instead of 4.2. Figure 2 shows the cumulative proportions contracting AIDS by year since infection, under two different sets of assumptions. Under high-threat assumptions, 95 percent of infected adults and children eventually contract AIDS. Under medium-threat assumptions, 50 percent eventually contract AIDS, though slightly earlier than under high-threat assumptions. Under either set of assumptions, the incubation period is much shorter for children than for adults. The rate of progression to AIDS among children implies that either 25 or 35 percent of those infected contract AIDS in their first year of life. This spread may bracket the possibilities. For instance, an Italian study of children of infected intravenous drug users showed 2 of 12 infected babies having contracted AIDS by six months (Semprini et al. 1987), implying a rate of at least 17 percent and possibly twice that or more in the first year.

The cumulative proportion having died from AIDS from the time of contracting the disease is defined by a separate logistic curve in Figure 2, which has k = 0.98, a = 0.85, and b = 1.00. Under both high-threat and medium-threat conditions, 98 percent of adults and children are assumed eventually to die, around 50 percent within one year of contracting AIDS. The rate was set to match reports of median survival after diagnosis of 11 months in the U.S.

These assumptions lead to reasonable scenarios, as comparisons with reported data show. Table 2 gives comparisons with Kinshasa data, with which model results mostly agree, and Table 3 gives contrasts, which are more difficult to evaluate, with U.S. estimates.

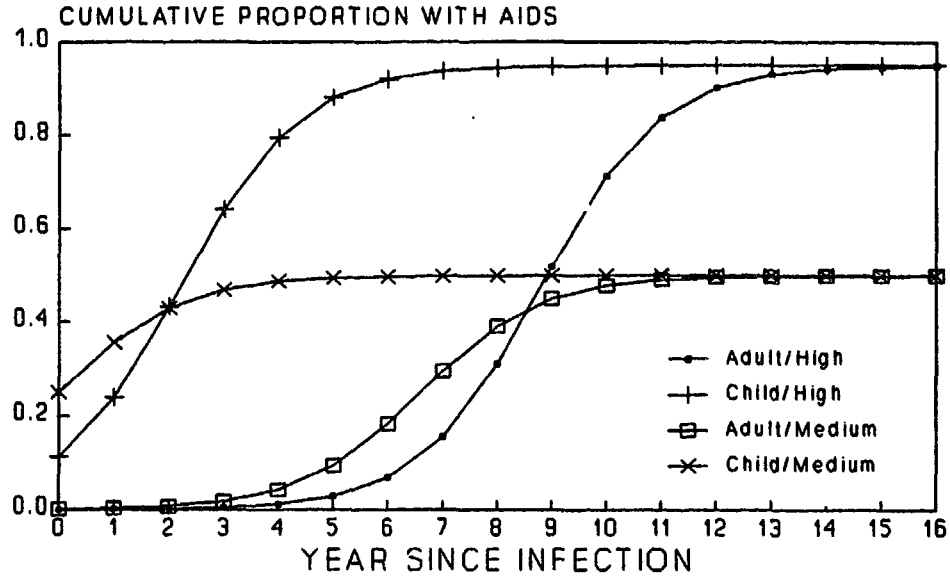
Two preliminary observations must be made, about the base for the model estimates and about the desirability of precise matching with reported data. Model rates in Table 2 have been calculated with urban adults as the base. Published data actually refer more narrowly to Kinshasa, which has a quarter of the urban population by the 1984 census. The distinction in this case is not consequential, however. If the simulation had started with an initial incidence rate based on Kinshasa adults, subsequent rates calculated on this narrower base would be virtually identical, assuming similar population growth between Kinshasa and all urban areas. Actual numbers of cases would of course be different.

Table 2 shows various model estimates generally close to reported rates. Tighter matching was not attempted because the reported estimates are based on such small samples that they cannot be considered precise. In addition, the model estimates shown are 1984-85 averages, to match the reports, and the change from 1984 to 1985 is substantial for some rates: for adult seroprevalence in the high-threat run, for example, from 0.027 to 0.034.

Adult seroincidence in the model is slightly higher than the reported estimate, though this may partly compensate for an underestimate among children. Seroprevalence levels, at any rate, and the speed with which seroprevalence grows, seem reasonably approximated. The latter involves a comparison with pregnant women, who are more likely to resemble the general population than prostitutes or men attending a sexually transmitted disease clinic. Studies of these other groups show far more rapid spread, but the groups are not representative. Pregnant women are not taken here as strictly representative either; otherwise, seroprevalence in 1985-86 would have to be set at 8 percent. Comparisons are made not with the precise dating of the spread in seroprevalence but essentially with the rate of spread.

The probability of progression to AIDS per year is reported at only 1.3 percent for adults both in a Kinshasa and a San Francisco sample (Mann et al. 1986c). The simulation model gives higher though not greatly higher rates, close to 2 percent, and still manages to match the predicted probability that 20 to 30 percent of current seropositives (in the U.S.) will contract AIDS

Figure 2 Cumulative proportions of infected adults and children with AIDS by year since infection, and cumulative proportion of AIDS cases of all ages having died by year since developing AIDS: Assumptions in high and medium threat runs



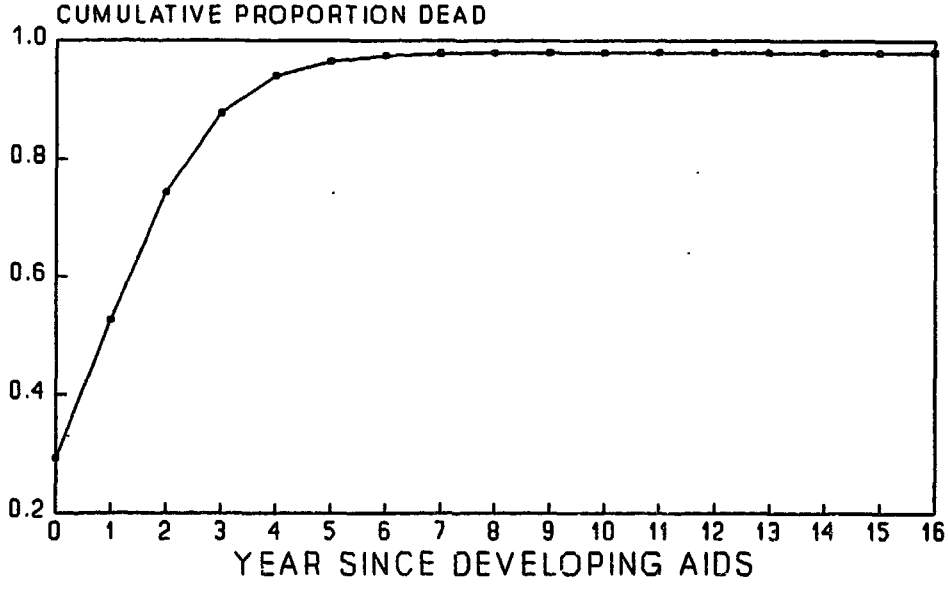


Table 2. Comparisons between reported and model estimates of HIV and AIDS incidence, prevalence, and deaths

Indicator and source of reported estimate (for Kinshasa, Zaire, except where indicated)	Reported	High-threat run	Medium-threat run
HIV infection incidence among adults 1984-85 579 seronegative hospital workers (Quinn et al. 1966)	.0075	.0081	.0078
HIV infection prevalence among adults 1984-85 5099 healthy persons (Quinn et al. 1986)	.030	.0304	.0290
Years for seroprevalence to grow from 0.2 to 3% 500 and 500 pregnant women (Quinn et al. 1986)	10.5	12.0	12.2
Sex ratio among infected by asymptomatic adults out of 5099 health persons (Quinn et al. 1986)	.66	.75	.75
Probability of contracting AIDS per year among infected but asymptomatic adults 67 cases enrolled in October 1984 (Mann et al. 1986c)	.0129	.0166	.0214
Probability of current HIV seropositives contracting AIDS in five years U.S. estimate (U.S. Public Health Service 1986)	.20-.30	.292	.263
Mean incubation period for adults in years 100 U.S. patients 13 years and older with transfusion-associated AIDS (Lui et al. 1986)	4.5	7.34	5.27
AIDS incidence per million adults, late 1984 surveillance data (Mann et al. 1986e)	380]	674	682
AIDS incidence per million adults, late 1984 "more reasonable estimate" (Quinn et al. 1986)	550-1000]		
Proportion of seropositives 0 years old out of 5099 healthy persons (Quinn et al. 1986)	.079	.062	.061
Proportion of seropositives 1 year old out of 5099 healthy persons (Quinn et al. 1986)	.012	.032	.027
Proportion of seropositives 2-14 years old out of 5099 healthy persons (Quinn et al. 1986)	.116	.060	.076
Median months of survival after contracting AIDS	11	10.7	10.7

Note: For the high-threat run, 95 percent of seropositives are assumed to eventually develop AIDS. For the medium-threat run, 50 percent of seropositives are assumed to eventually develop AIDS.

within five years. (The model estimates shown are actually for 1980 seropositives contracting AIDS by 1985.) It is relevant to ask how these estimates can be consistent. The explanation is that the base for the estimate of the annual probability of contracting AIDS excludes adults with generalized lymphadenopathy, an early indication of infection, distinct from frank AIDS, that develops in 9.9 percent of asymptomatic seropositives each year (Quinn et al. 1986).

Assuming a Weibull distribution, Lui et al. (1986) estimate a mean incubation period for HIV of 4.5 years, with a 90 percent confidence interval of 2.6 to 14.2 years. Through indirect methods that depend on the basic biology of HIV, May and Anderson (1987) produce a rough estimate that this period exceeds 5 years. The simulation model deliberately adopts an average incubation period one to three years longer, which is still within Lui and his coworkers' confidence interval and close to estimates they make using alternative distributions. Changes in the parameters to reduce the incubation period appear to lead to too high an annual probability of contracting AIDS.

Infants with AIDS are slightly underestimated in both simulation runs. The most likely explanation is that substantial numbers of infants are infected through injections or blood transfusions (Mann et al. 1986b). Infants are at risk from infected needles or blood whether their mothers are seropositive or not. Such infections, which appear to account for 17 to 40 percent of AIDS cases among children in a Kinshasa hospital, are a partial justification for allowing a perinatal transmission rate of 0.70 rather than lower. In earlier stages of the epidemic, when seropositive women are relatively few, these sources of infection could be more important, but should decline in relative importance over time as seropositive mothers increase and as doctors and nurses take more precautions.

The reported proportion of seropositives aged 1 is overestimated in the model. A very rapid progression to AIDS is necessary to match the reported cut (Table 1) in the number of healthy seropositives of 85 percent between the first and the second year of life. On the other hand, the proportion of seropositives aged 2 to 14 is underestimated, probably because of children infected through needles or blood.

No simple solution is available to make the model fit the data on child seropositives more exactly. A higher transmission rate might be used, but this appears unrealistic and, while allowing matching of current estimates, would produce an upward bias in projections for the future. Nor is it clear whether infants and children infected through needles or blood contract the disease and die from it at the same rate as children infected at birth. The U.S. National Academy of Sciences (1986:298) in fact reports that, while children infected at birth are "usually symptomatic by six months of age," children infected through transfusions during the first year of life "may be clinically well from one to four years." Treating these children together with adults is not an adequate solution either, because they are not involved in the sexual transmission of the virus. Other than allowing adult seroincidence to be slightly higher than reported, no further attempt at a closer fit to the data was made.

Table 3 compares simulation results to estimates for the U.S. What ratio to expect between numbers of cases in Zaire and the U.S. (assuming similar processes of spread) is difficult to say a priori: numbers could arguably be roughly equal, or numbers could be in proportion to population. No overall conclusion can be drawn from this table.

The model does appear to produce more cumulative AIDS cases and deaths relative to current infections, new AIDS cases, and current deaths than are estimated for the U.S. The possible changes in parameters for transmission and progression of the disease that would keep the simulations consistent with Kinshasa data would not greatly alter the balance between

Table 3. Comparisons between reported estimates of AIDS for the U.S. and model estimates for Zaire

Indicator	U.S.--Public Health Service estimates	Zaire--model estimates (000)		Ratio: Zaire/U.S. estimates	
		High-threat run	Medium-threat run	High-threat run	Medium-threat run
Infection prevalence 1985	1-1.5 m.	219	209	.17	.17
Cumulative AIDS cases to 1984	19,000	13.8	14.7	.73	.77
New AIDS cases 1985	16,000	4.9	4.9	.30	.31
Cumulative surviving AIDS cases to 1984	10,000	5.7	5.8	.57	.58
Cumulative deaths to 1984	9,000	10.3	11.1	1.15	1.23
Deaths in 1985	9,000	3.7	3.7	.41	.42

Note: The population of Zaire is approximately 13 percent of the U.S. population. The U.S. estimates are from the Public Health Service (1986).

cumulative and current cases. A later start of the epidemic in the U.S. is one possible explanation for the difference in the balance between cumulative and current cases, but other differences in the dynamics of the epidemic may also be important explanations.

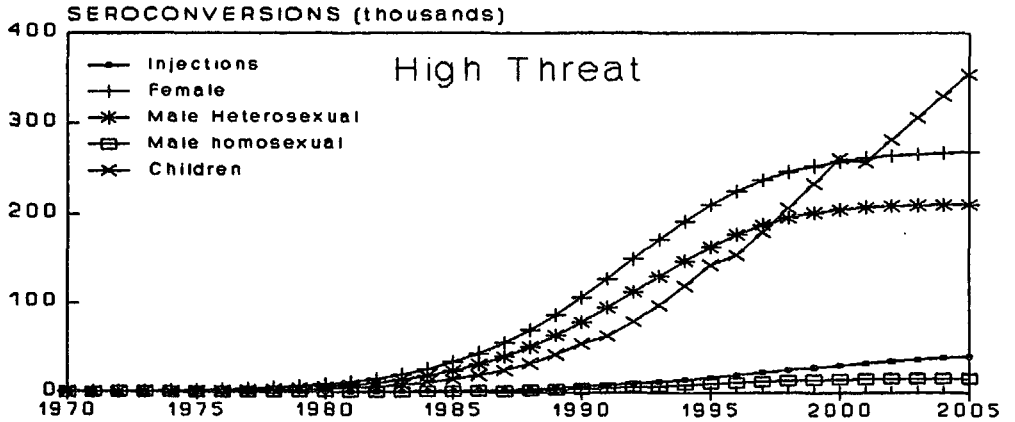
Projection

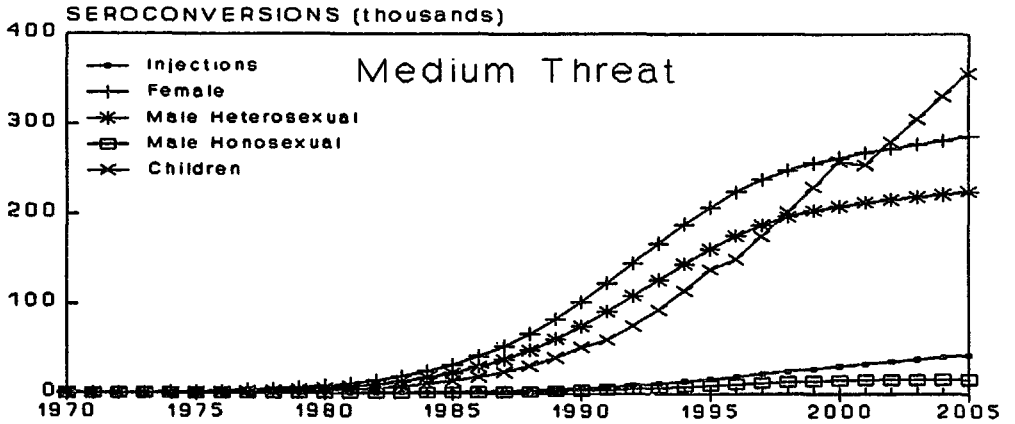
Four population projections were run for Zaire: a standard projection with no effect of AIDS; a high-threat run; a medium-threat run; and a declining-threat run.

The medium-threat assumptions are adopted for the declining-threat run, except that various transmission parameters are allowed to change. In the area of sexual behavior, unprotected sex acts in each risk group are assumed to decline 5 percent a year. Number of new sexual partners is reduced 1 percent a year in each high and medium subgroup, but does not change in each low subgroup. Faster change is allowed for injections. The proportion of needles sterilized rises 8 percent a year, reaching 0.93 after 20 years; and the number of injections falls 5 percent a year in the high subgroup, but is unchanged in the other two subgroups. The perinatal transmission rate drops 2.4 percent annually, making it 0.55 after ten years and 0.43 after 20 years. The assumption here is that infection of children through needles or transfusions will eventually be controlled, but maternal infection will be less tractable.

Figures 3 to 6 show some projection results for the three runs incorporating AIDS. The high-threat and medium-threat runs generate similar estimates of seroincidence and seroprevalence. Annual seroincidence among adult women reaches 275,000 in 2005, while seroincidence among adult men is slightly lower. For both sexes, seroincidence shows a tendency to level off. For children, however, seroincidence may still be accelerating by 2005, when it has reached 350,000. (The curves for children are not smooth because of discontinuous changes in

Figure 3 Annual HIV seroincidence among adult injection recipients, females, heterosexual males, homosexual males, and children: Projections under high, medium, and declining-threat assumptions





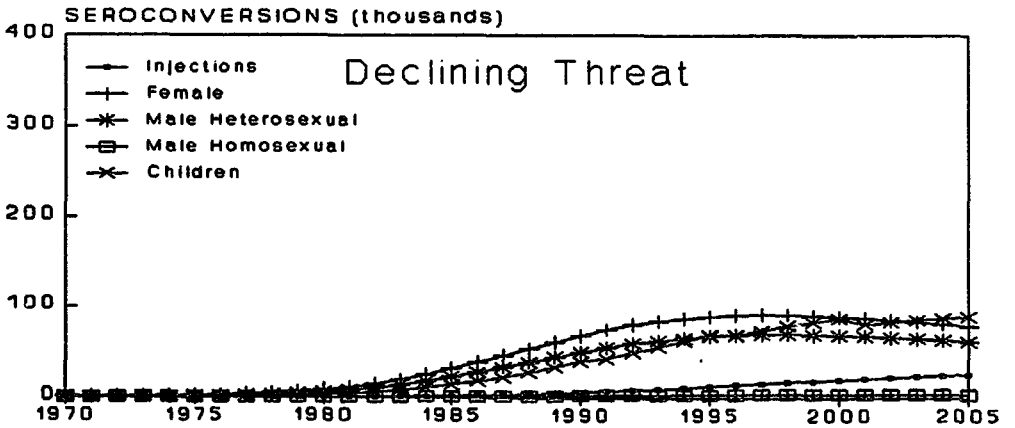


Figure 4 HIV seroprevalence among adults and children by year: Projections under high, medium, and declining-threat assumptions

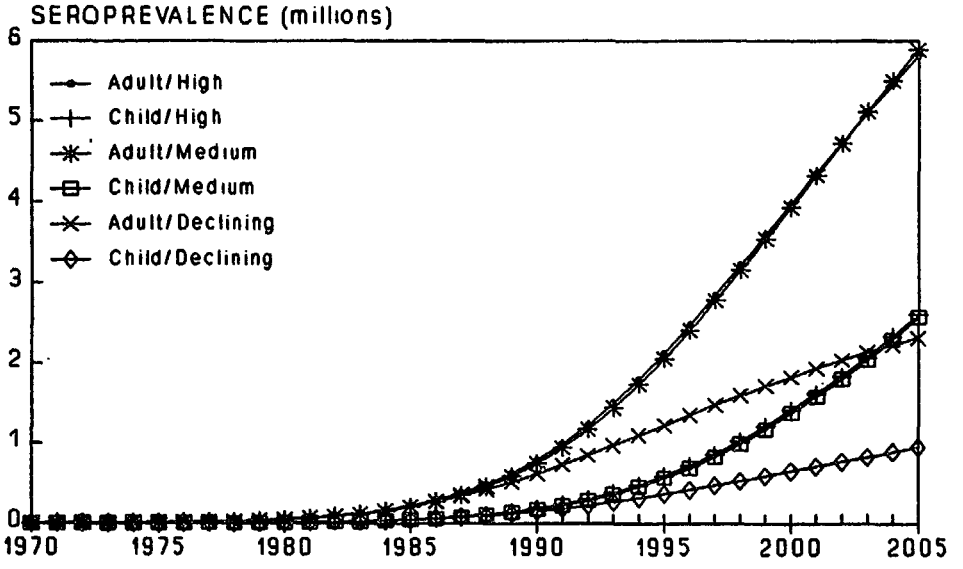


Figure 5 Annual incidence of AIDS among adults and children: Projections under high, medium, and declining-threat assumptions

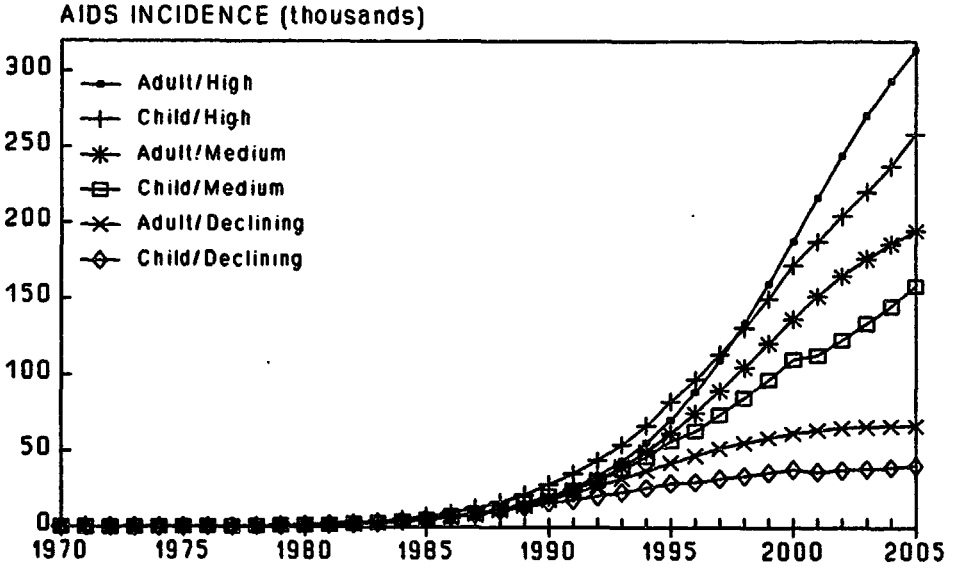
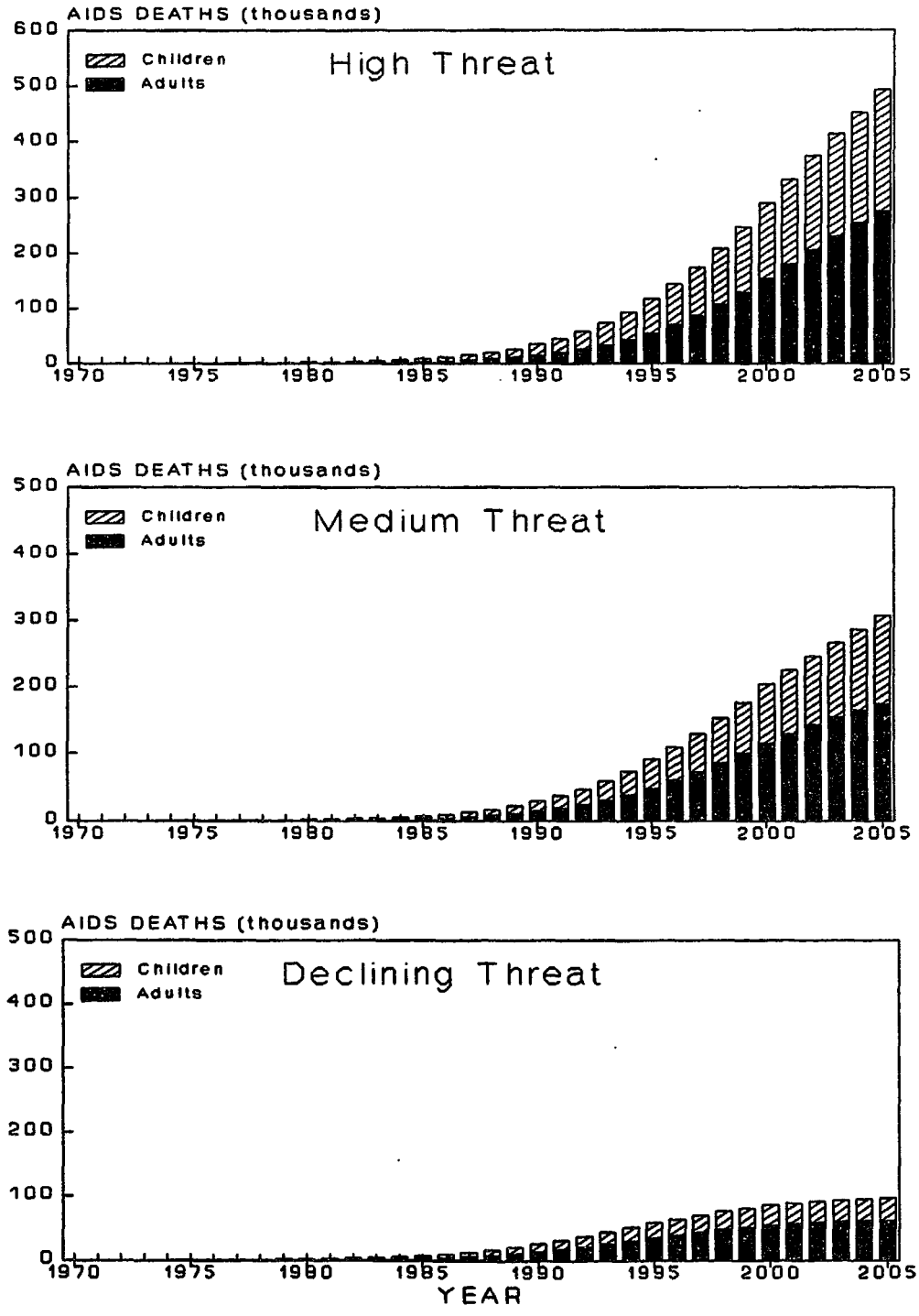


Figure 6 Annual deaths from AIDS among adults and children: Projections under high, medium, and declining threat assumptions



fertility.) The declining-threat run produces much lower seroincidence by 2005: 80,000 annually for women, 65,000 for men, and 90,000 for children.

Seroprevalence is of course much higher, close to 6 million adults and 2.5 million children by 2005 in both high-threat and medium-threat runs. In the declining-threat run, the numbers are much smaller but still substantial: 2.3 million adults and 1 million children.

Because of the long incubation period, AIDS cases are many fewer. New cases in 2005 are only one-half to two-thirds of the number of seroconversions in the same year. Deaths from AIDS are still slightly fewer than new cases. The contrasts between the runs are stark, however: deaths in 2005 are 500,000 in the high-threat run, 300,000 in the medium-threat run, and 100,000 in the declining-threat run. The number of deaths also appears to be levelling off in the declining-threat run, but not in the other runs.

The impact on population growth and mortality rates is shown in Table 4 and Figure 7. Population is virtually unaffected in all three runs, falling at most 3 percent short of what it would be without AIDS. The increase in life expectancy expected from improvements in levels of living and medical care does not take place under high-threat assumptions; life expectancy instead is virtually constant. Under medium-threat assumptions, the increase in life expectancy over 15 years is only half what it would be otherwise. Under declining-threat assumptions, life expectancy does improve more, falling 3 percent short of the standard projection. The infant mortality rate is strongly affected: instead of falling to 70 in 2000-2005, it rises to 130 under high-threat assumptions. Because child and adult deaths balance each other out, the dependency ratio is hardly affected.

The impact of AIDS on population in these runs comes more from its effects on children than its effects on adults. The incubation period among adults is sufficiently long to allow many of them to reproduce, and their deaths occur after the losses have in effect already been made up. This is not the case for infected children, most of whom die, in all three runs, before reaching the age of five.

DISCUSSION

These results depend on the structure of the model and on the parameters chosen for each run. Like any model relating to social behavior, this one does not capture the full complexity of the phenomenon. Of the many possible reasons for skepticism about the validity of the results, 37 will be noted under three headings: limitations in the model itself; uncertainties about the values used for the parameters; and inherent problems with projections of social behavior. Areas for further work will also be mentioned.

Model limitations

As operationalized, the model is difficult to expand, and it cannot accept much more complexity. This accounts for some of its limitations, though not all. Limitations will be considered in six areas: general; transmission generally; sexual transmission; nonsexual transmission; progression to AIDS and death; and linkage with demographic projections.

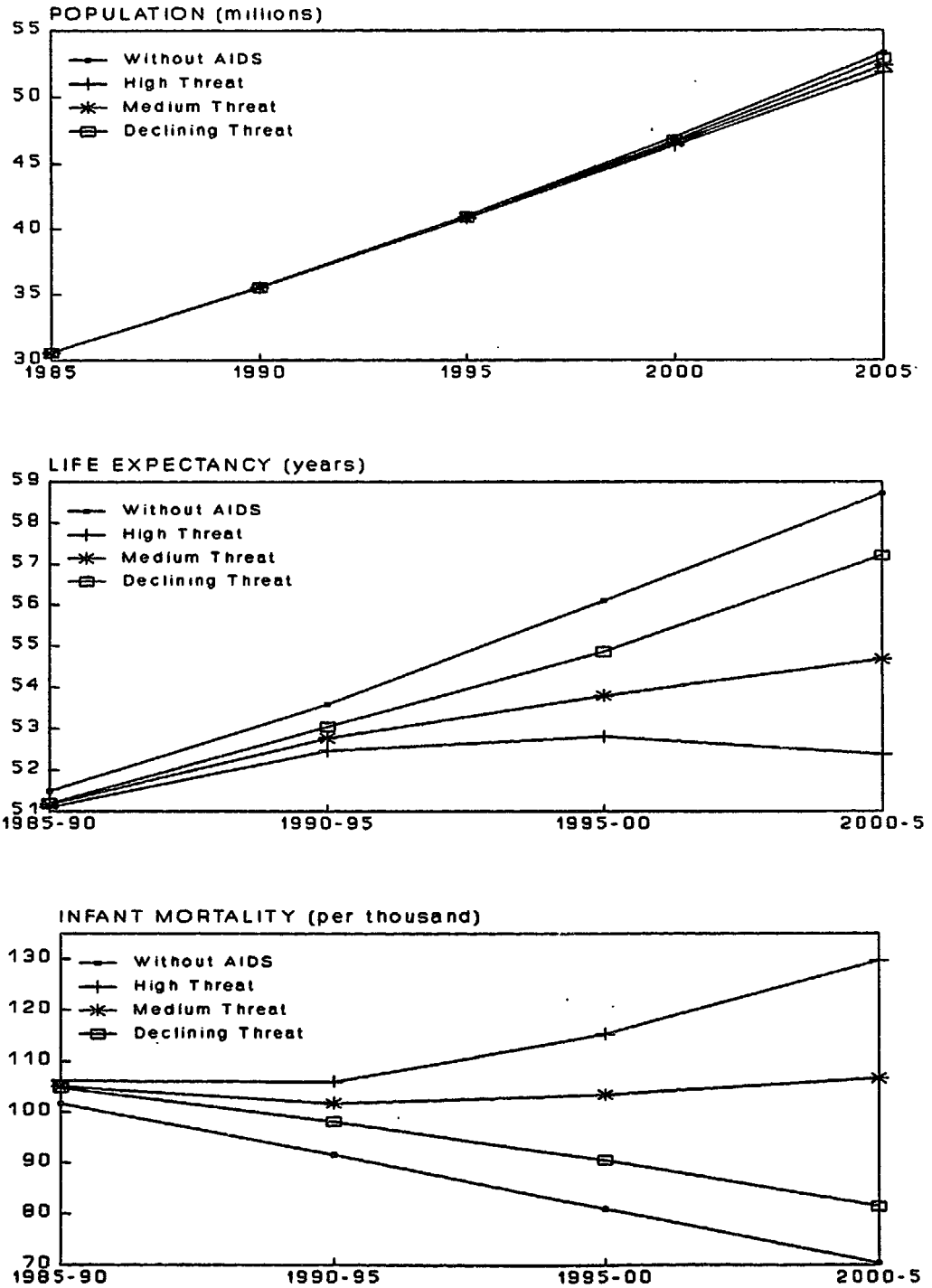
General

1. The model recognizes only one virus as causing AIDS. A second virus, HIV-2, has now been identified, and the possibility that other viruses exist is also open. For some countries, a two or three-virus model may eventually be needed, but so little information exists about HIV-

Table 4. Standard population projection without AIDS and three alternative projections with AIDS: Zaire, 1985-2005

Year	Standard, without AIDS	With AIDS: Degree of threat			As proportion of standard: Degree of threat		
		High	Medium	Declining	High	Medium	Declining
Population							
1985	30557	30557	30557	30557	1.00	1.00	1.00
1990	35560	35516	35525	35527	1.00	1.00	1.00
1995	41001	40830	40878	40915	1.00	1.00	1.00
2000	46957	46367	46546	46712	0.99	0.99	0.99
2005	53298	51842	52338	52851	0.97	0.98	0.99
Growth rate							
1985-90	3.03	3.01	3.01	3.01	0.99	0.99	0.99
1990-95	2.85	2.79	2.81	2.82	0.98	0.99	0.99
1995-00	2.71	2.54	2.60	2.65	0.94	0.96	0.98
2000-05	2.53	2.23	2.35	2.47	0.88	0.93	0.98
Death rate							
1985-90	14.6	14.9	14.8	14.8	1.02	1.01	1.01
1990-95	13.3	14.0	13.8	13.6	1.05	1.04	1.02
1995-00	11.6	13.5	12.9	12.3	1.16	1.11	1.06
2000-05	10.0	13.4	12.1	10.7	1.34	1.21	1.07
Life expectancy at birth							
1985-90	51.5	51.1	51.2	51.2	0.99	0.99	0.99
1990-95	53.6	52.5	52.8	53.0	0.98	0.98	0.99
1995-00	56.1	52.8	53.8	54.9	0.94	0.96	0.98
2000-05	58.7	52.4	54.7	57.2	0.89	0.93	0.97
Infant mortality rate							
1985-90	102	106	105	105	1.04	1.03	1.03
1990-95	92	106	102	98	1.16	1.11	1.07
1995-00	81	115	104	91	1.42	1.28	1.12
2000-05	70	130	107	81	1.84	1.52	1.16
Dependency ratio							
1985	93	93	93	93	1.00	1.00	1.00
1990	93	93	93	93	1.00	1.00	1.00
1995	91	91	91	91	0.99	1.00	1.00
2000	89	87	88	88	0.99	0.99	1.00
2005	80	80	81	82	0.99	1.00	1.01

Figure 7 Total population, life expectancy at birth, and infant mortality rate, 985-2005: Projections without AIDS and under high, medium, and declining-threat assumptions



2 (which appears to differ in pathogenicity from HIV-1) that parameterizing such a model is not possible at this time. For the present case, HIV-2 has not been reported as a major factor in Zaire.

2. The model uses fixed rather than statistically variable parameters. No estimates are possible, therefore, of the variance in the results.

Transmission, general considerations

3. Seroconversions are calculated year by year, in effect allowing only the previous year's seropositives to infect others this year. Those infected cannot go on to infect others until the succeeding year. This slows the apparent spread of HIV, particularly among the most sexually active groups. The model may therefore give an underestimate for the early stages of the epidemic, and possibly an overestimate for later stages.

4. The model does not provide for genetic variables that may affect likelihood of infection (Eales et al. 1987). If such variables are important, the model probably overestimates the impact of AIDS in the long run.

5. The model assumes all seropositives are equally infective, excluding those who have developed AIDS, who are assumed to be sexually inactive. Recent reports indicate, to the contrary, that seropositives increase in infectivity as the disease progresses. Taking this into account would probably imply an acceleration in the rate of seroconversion at some point.

6. Whether reinfection by HIV is possible, and whether it accelerates progression to disease, does not appear to be resolved. The model allows an individual to be infected only once.

7. The potential effect of vaccines is not directly allowed for, although appropriate reductions in transmission parameters might be chosen to approximate vaccine impact. Since vaccines are reported to be on the order of a decade or more away, and since they are unlikely to be immediately used throughout the developing world, ignoring their potential effect at this point is not inappropriate.

Sexual transmission

8. Bisexuals are not incorporated into the model. Given that homosexuals appear to be a minor risk group in Zaire, bisexuals are probably not a significant factor in the present case.

9. A key assumption in this model is "proportionate mixing" (Hethcote and Yorke 1984), the idea that every pairing of sexually active individuals is equally probable, if their activity rates are taken into account. This assumption is certainly untrue but is difficult to avoid, and therefore underlies various models for sexually transmitted diseases. The segmentation of individuals into separate, minimally interacting, sexual "markets" probably implies slower spread than the model represents.

10. The focus is on new sex partners in order to exclude stable marital or quasi-marital relationships into which neither partner is likely to introduce the virus. But people do get infected by old sex partners who have themselves had new partners or been infected through other means. How to deal with this possibility is not obvious.

11. The model does not require that the new sex partners of heterosexual women in a given year be equal to the new sex partners of heterosexual men. Parameters for number of new

partners can therefore be set inappropriately. What effect this has depends on how unbalanced the parameters chosen are.

12. Number of new sex partners per year and number of unprotected sex acts per partner are assumed to be constant over an individual's lifetime. In reality, these numbers are highest among young adults and decline in later years. The model's inability to take this into account does not affect the age distribution of adult deaths (which is separately determined), but may have other effects difficult to foresee.

13. Anal sex is apparently more effective at transmitting the virus than genital sex; the effectiveness of oral sex is undetermined. These variations are ignored, except to the limited extent of allowing differential transmission among homosexuals.

14. Anogenital ulcers facilitate virus transmission. The only way in which this is taken into account in the present case is to allow a slightly higher transmission rate than that reported in the U.S.

15. The effectiveness of condoms at preventing transmission may be about 90 percent, though it is not clear how consistent is the pattern of condom usage that underlies this estimate. The model assumes that condom usage completely prevents transmission. If it is desirable to assume 90 percent effectiveness instead, the number of unprotected sex acts could be increased by adding 10 percent of the acts in which condoms are used.

Nonsexual transmission

16. Injections and transfusions are not distinguished. In some cases, this limitation might be overcome by careful choice of parameters relating to injections-transfusions.

17. A more serious limitation is the model's inability to include infections of children through injections and transfusions. For the present case, the extent to which a higher perinatal transmission rate makes up for this is unknown.

18. The process by which needles get infected needs better specification. The proportion of needles infected bears some relationship to the number of seropositives in the population. The model says that the proportion of needles that carry the virus depends on the number of seropositives and the frequency with which they receive injections. No more accurate description of what actually happens has been identified.

19. Intravenous drug use is a major means of transmission in developed societies. Its importance in many developing societies is unknown. The model makes no specific provision for intravenous drug use, which becomes in effect another aspect of injections-transfusions. Since drug users are a special population quite different from the population that receives frequent injections for other reasons, this lumping together of different groups can complicate interpretation of results.

Progression to AIDS and death

20. Logistic distributions have been used, for convenience, to represent the incubation period and the period between diagnosis of AIDS and death. The choice of a normal or a Weibull distribution instead would not appear to make significant difference. Nevertheless, it should be noted that no firm basis exists for the choice of a distribution.

21. Progression to disease and death is assumed to be uniform, except for the distinction

between adult and child progression. Recent work has begun to suggest that progression may in fact be faster among older people, in their late thirties, than among younger adults. Progression may also be affected by genetic differences. Cofactors like these have not been taken into account.

22. Deaths due to HIV infection are all assumed to take place only after the individual contracts AIDS. No deaths are assumed to take place because of aids-related complex (ARC) or other conditions apart from frank AIDS.

23. Azidothymidine (AZT) and a number of other drugs hold out the possibility that the life of AIDS patients may be extended, and possibly the development of AIDS postponed. Restoring immune function, however, appears to be a more distant goal. The model does not allow for the effect of such drugs, but their limited availability, cost, and side effects will prevent their widespread use in developing countries in the near future.

24. Other conditions related to HIV infection, like generalized lymphadenopathy, ARC, and HIV neurological disease are not represented in the model. The excess morbidity due to the AIDS pandemic is therefore underestimated. To the extent these conditions are life-threatening, mortality may also be slightly understated, though mortality effects could in principle be handled by grouping more serious conditions with AIDS itself.

Linkage with demographic projections

25. Calculations relating to children are age-specific, but calculations for adults are not, except that an age distribution of the asymptomatic is applied to give an age distribution of deaths. It would be more precise to make the model keep track of the specific age distribution of adult seropositives, though this would introduce considerable complexity and require more age-specific data.

26. Deaths from AIDS are not considered to affect mortality rates until the 15-year simulation period is succeeded by the 20-year projection period. This limitation is not serious for two reasons. First, deaths in the early part of the epidemic are so few that they fall well within the limits of uncertainty about actual mortality rates. Second, one could if desired shorten or eliminate the simulation period.

27. The spread of AIDS is assumed to have no effect on fertility or on mortality from other causes. Regarding mortality, an increase in deaths from other causes among HIV seropositives could simply be counted as additional deaths due to AIDS. However, serious infectious diseases that HIV seropositives spread to seronegatives are more difficult to handle. For fertility, similarly, no easy fix is possible--but to date no data are available about how fertility behavior will respond.

28. The model allows only seronegatives to migrate in or out of the society. If future migration trends could be predicted, the model might require correction in this regard. But future trends are difficult to predict in any case. In addition, the more important factor in the spread of HIV is temporary movement, for business, tourism, and other reasons. This is not counted here, as it almost never is in population projections.

Uncertain parameters

The values of some parameters for the model were chosen based on scanty and clearly inadequate data. The values for others, on the other hand, were simply made up. Major uncertainties relating to the parameters used will be noted. Though many uncertainties exist, they

are certainly fewer than would exist if the model were elaborated to take the limitations noted above into account. The three major areas of uncertainty are social, epidemiological, and demographic.

Social

29. Reliable, representative data on sexual behavior are virtually nonexistent. Studies of AIDS cases have begun to include some information, but the biases in such data are difficult to overcome. Therefore, the values used here were all made up, their only justification being that they appear to give reasonable results in the simulation period.

30. Behavior relating to injections appears to be no better understood. In this case again, invention of parameters was the daughter of necessity.

Epidemiological

31. Accumulating studies provide data on perinatal transmission and transmission in sexual acts. More of these studies refer to developed than to developing countries, however, and the figures they provide do not necessarily correspond to model parameters.

32. If data were available on the proportions infected through different means, calibration of a model would be considerably facilitated. Whereas some studies identify cofactors for infection, it is not possible to tell, most of the time, what weights should be given to each factor.

33. Estimates of HIV seroprevalence and seroincidence and AIDS incidence were available for Kinshasa and were essential in this exercise. Undoubtedly these estimates are not as firm as they should be: one estimate of AIDS incidence, for instance, is double to triple another. Also essential for the exercise was an age-sex distribution of seropositives. The sample in this case may or may not be representative. Even in this area, therefore, where more data appear to exist than elsewhere, uncertainties in the figures indicate the need for caution in interpreting results.

Demographic

34. As noted earlier, population estimates for Zaire are not fully adequate and require much more analysis. Age-sex structures are not available and have been taken from models for this exercise.

35. Similarly, information on fertility, mortality, and migration in Zaire needs further analysis to determine best estimates.

Projection limitations

Instead of simple extrapolations, the projections here rely on a complicated apparatus to carry experience of the past into the future. By identifying the areas in which guesses about the future are made and providing a structure within which the impact of these guesses can be worked out, projections do sometimes provide better insight than simple extrapolations. Nevertheless, these projections rely at root on guesses about future behavior, and therefore require qualifications beyond those that result from model limitations and parameter uncertainty.

36. Transmission parameters are assumed to remain unchanged in two of the runs and to change in a uniform pattern in the last run. Even if these parameters can be assumed to be properly specified, it is unlikely that they will remain unchanged or change in exactly the fashion specified. Changes in sexual behavior in the U.S. might have been used to define expected changes

in the projections, but circumstances and initial parameters probably are sufficiently different so that this would not be a good guide.

37. No better known are future trends in fertility, mortality, and migration. They have been specified here using standard World Bank methodology rather than from any specific consideration of circumstances in Zaire. Considerable uncertaintly therefore attaches to the time path of vital rates.

One feature of the projections that might suggest a limitation is the fact that they indicate little impact on population despite the large effect on morbidity and mortality. This can, however, be explained in this fashion. The impact on population size and growth depends not only on the numbers affected but on timing. For this disease, the latency period appears sufficiently long to lead to minimal effect. Especially in a rapidly growing population, the deferred losses to AIDS may in effect have been made up in advance. The lapse of close to a decade between infection and death substantially reduces the impact on population. Without declines in transmission, however, serious effects on population size may still be possible beyond the projection period.

Further work

Further work is needed in many areas, as the preceding list indicates. Alternative, more complex models are needed if more features of the pandemic are to be taken into account. Developing such models is one priority, but another is to use the current model to answer various questions, such as these:

o Does the demography make a difference? Would a lower-fertility population, like the U.S. population be less strongly affected? Similarly, how would the impact of the disease be reduced by greater decline in fertility?

o How would the spread of the disease be different if sexual behavior changed differentially across groups? Can a substantially greater effect be confirmed if those most sexually active change their behavior first?

o How much difference in the spread should be expected where transmission is primarily or largely homosexual? Fewer children are likely to be affected initially, and this could produce quite different demographic results.

o How substantial would be the reduction in the magnitude of the epidemic if transmission declined earlier? For countries at earlier stages of the epidemic, this question may be critical.

o Can economic costs be assigned for the disease based on these projections? Much additional data and many more assumptions would be necessary, but in principle an extension of the exercise might be possible.

APPENDIX: THE INCUBATION PERIOD

Two alternative distributions for the probability of developing AIDS, given the date of infection, have been proposed. Neither is entirely satisfactory for the present exercise. An alternative distribution is discussed.

Lui et al. (1986) and Rees (1987) fit a Weibull distribution and a normal distribution, respectively, for the probability of developing AIDS over time to essentially the same data, on

transfusion-associated AIDS in the U.S. These data, though they have various limitations, allow precise determination of the date of infection.

Lui et al. do not give the equation they estimated, though they do give a graph of what they label the probability density. This graph clearly implies that fewer than 15 percent of HIV seropositives--perhaps as few as 10 percent--eventually develop AIDS. This implication is inconsistent with the current view that at least 50 percent of seropositives in fact progress to AIDS.

An attempt was made to reestimate the curve by minimizing the likelihood ratio statistic

$$G^2 = 2 \Sigma O \ln (O / E)$$

O stands for observed frequencies of AIDS cases by infection cohort and year since infection, taken from Rees. (Following Lui et al., observations prior to 1982, the date of the first diagnosis of transfusion-associated AIDS, were eliminated. Some cells have zero frequencies; this was changed to 0.01 to allow computation of G^2.) E stands for expected frequencies, determined by distributing the total number of cases in each cohort to match that portion of the density function covering the period over which the cohort was observed. The resulting equation is

$$w(t) = 6.8 (30)^{-6.8} t / \exp [(t / 6.8)^{30}]$$

with a likelihood ratio statistic of 19.75. The mean of this distribution is 4.5, identical to Lui et al.'s estimate. Similarly, this density function, if considered a probability density, implies that extremely few seropositives contract AIDS. It also has an unusual shape, increasing erratically until t is around 6 and then falling precipitously to zero by the time t reaches 7.2.

Rees concludes, on the other hand, that a normal distribution with a mean of 15 years and a standard deviation of 5 years fits the data better than any other normal distribution. This mean is unusually high, falling outside the 90 percent confidence interval for Lui et al.'s estimate and also differing by an order of magnitude from an indirect estimate by May and Anderson (1987).

Parameters for a normal distribution were reestimated following the same procedure as for the Weibull. The resulting estimates were a mean of 5.0 and a standard deviation of 2.0. (The likelihood ratio statistic is 22.49.)

In fitting a distribution, the main problem lies with the right-censoring in the data, which exclude all cases of infection that had not led to AIDS as of the reporting date. The distribution of AIDS cases beyond this date is unknown. More critically, the numbers infected in each year are also unknown. As a consequence, no sound basis exists for inferring the proportion of seropositives who develop AIDS in any given period since infection; one can only infer the ratios of such proportions across periods (Barton 1987). In other words, one can extract from the data the shape of part of a probability density function, but one cannot tell how large or how small a part of the curve is represented. Applying these distributions leads to inference of the possibly substantial remainder of the curve from the shape of the available part.

An alternative approach is to fit a distribution with some parameters dictated by the censored data but at least one other parameter independent of the data, and therefore variable at will to represent alternative possibilities in areas where the true values are unknown and cannot be safely inferred. Such a distribution might be obtained by multiplying a Weibull or a normal by an additional parameter k. Instead, an alternative distribution is used here, a logistic distribution of the form

$$L(t) = k_0 + k / [1 + \exp (a - bt)]$$

with k_0 set to zero. The equivalent density function is

$$l(t) = k\ b\ e^{a-bt} / (1 + e^{a-bt})^2.$$

The parameter k conveniently gives the proportion of all seropositives who eventually develop AIDS, but its value is irrelevant in fitting the curve.

The parameters a and b were estimated by the same procedure discussed above for the Weibull distribution. The resulting estimate for a is 4.2 and for b is 0.91, with a likelihood ratio statistic of 25.12. Observed and expected frequencies are compared in Table A. The estimated frequencies are not affected by changing the value of k, and neither is the mean--which equals 4.5 years, close to estimates from the other distributions. The differences among these distributions (provided the Weibull and the normal are multiplied by an equivalent parameter k) therefore appear relatively minor.

Table A. Observed frequencies and frequencies expected from a logistic distribution of AIDS diagnoses by infection cohort and years since infection, transfusion-related AIDS in the U.S.

Years since infection	1978		1979		1980		1981		1982		1983	
	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp
0.5							1.0	2.0	3.0	4.6	4.5	7.5
1.5					0.0	2.1	7.5	4.7	14.5	10.7	20.5	17.5
2.5			2.0	1.9	8.0	4.5	8.5	9.9	20.5	22.7		
3.5	0.0	0.7	1.0	3.3	11.5	7.9	17.0	17.4				
4.5	0.0	0.9	5.5	4.2	5.0	10.0						
5.5	2.0	0.8	4.5	3.6								
6.5	1.0	0.5										

The estimated means from the different distributions may all be low relative to the true mean for all AIDS cases, because these patients could have been infected with substantially larger doses of the virus through transfusion than others receive through sexual contact. Since they required transfusions, they may also have been initially debilitated or subject to other infections and more likely to develop AIDS quickly.

Much more could be learned about the incubation period and how it varies if data were also available on an appropriate sample of seropositive transfusion recipients, with no other risk factors, who have not developed AIDS. Without such data, any conclusions about the incubation period must be purely tentative.

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IMPLICATIONS OF CONTROL MEASURES FOR THE SPREAD OF HIV INFECTION

Rodolfo A. Bulatao and Eduard Bos

Abstract. The effects of changes in sexual behavior and of medical interventions on the spread of Human Immunodefiency Virus (HIV) infection are illustrated using an epidemiological-demographic model for the disease. Parameters were chosen for the model to represent an African country with relatively high HIV seroprevalence, high fertility, and high mortality. Condom use is the most effective of the interventions assessed. Reducing genital ulcers, providing clean needles, and cleaning the blood supply have minor effects. Interventions have similar impact regardless of HIV seroprevalence level and level of sexual activity.

No control measures will be adequate to prevent many infections and deaths from the Human Immunodeficiency Virus (HIV) in the next two decades, but changes in sexual behavior could moderate the spread of the epidemic. Available "medical" interventions, on the other hand, will have minimal effect. This is demonstrated using a model that simulates the spread and progression of the disease in an African country with relatively high HIV seroprevalence.

Then the question of the settings in which control measures might have more effect is tackled. It is shown that similar patterns of condom use will have equivalent effects despite different levels of sexual activity or different levels of HIV seroprevalence.

METHOD

First, we discuss a simple representation of possible control measures and their effects, and then we briefly characterize the simulation model used to assess these effects.

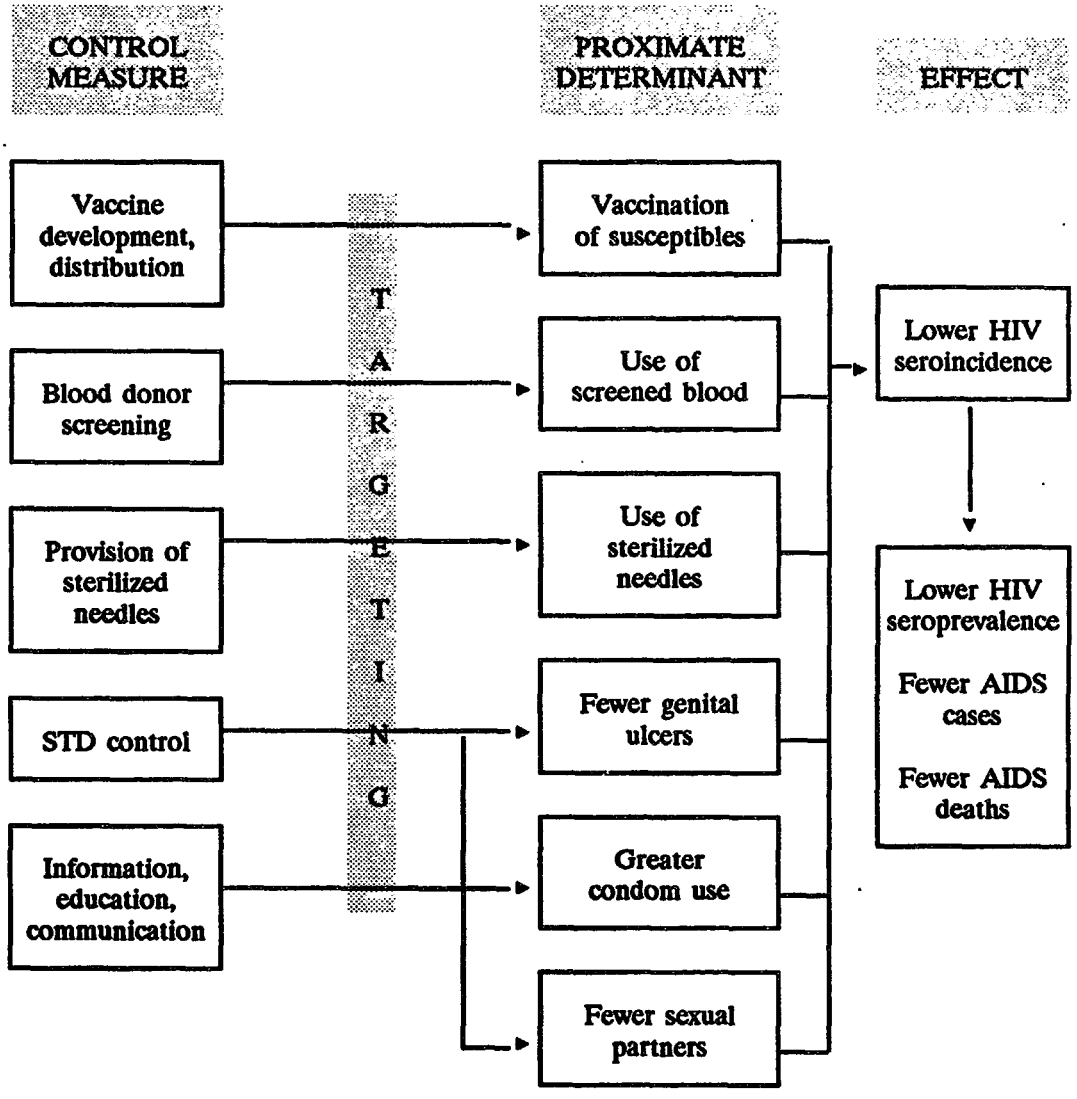
Figure 1 includes five possible control measures, from vaccine development to information, education, and communication (IEC) programs. Each measure gives rise to particular behaviors in the population, these behaviors being the proximate determinants that lead to modifications in HIV transmission. Figure 1 suggests that targeting control measures on different groups may lead to important differences in the distribution of the proximate determinant behaviors across the population.

How the control measures affect the proximate determinants is poorly understood. For instance, IEC is known to increase condom use, but what types of IEC, how much of it, and how much must be invested in it are all questions without reliable answers. Empirical research is seriously lacking on these issues. Therefore, we focus here only on the proximate determinants and their impact on the HIV epidemic.

We also exclude consideration of the effects of a vaccine, since development may be a decade or more away, and distribution would impose further delays. When realistic prospects for a vaccine develop, this exclusion can be reconsidered.

The simulation model is a standard epidemiologic-demographic model that allows HIV to be transmitted sexually, perinatally, through needles, or through blood, represents the progression from infection to frank AIDS mainly from data on transfusion cases, and projects the population

Figure 1 Effects of control measures on the HIV epidemic



to provide estimates of susceptibles. The model resembles that described in the preceding note but is more complex in a number of ways. For instance, it allows infectivity to vary with duration since infection.

Parameters were obtained for the model from the literature and, where this did not suffice, were estimated iteratively by running the model and attempting to fit reported rates for a Central African country with relatively high HIV seroprevalence (as was done in the previous note).

A base scenario was constructed, in which all the proximate determinants were assumed to stay constant from the present to 2010. Alternative scenarios were then constructed as variations on the base scenario, allowing the proximate determinants to vary individually, increasing or decreasing by some constant percentage beginning in the year 1990.

RESULTS

Effects of changes in sexual behavior

Figure 2 illustrates the assumed trends in sexual behavior, and Figures 3 and 4 show their effects. Condom use (assumed to be 90 percent effective in preventing infection) is represented by alternative scenarios in which the percentage not using condoms declines annually by either 2 or 10 percent. (The rate of change is applied to nonusers rather than users in order to allow slower growth in condom usage as the proportion using approaches high levels and to prevent this proportion from exceeding 100 percent.) These rates of change resemble rates of change for nonuse of all contraception: Kenya, for instance, achieved a 2 percent annual reduction in contraceptive nonuse between 1977 and 1984, Mauritius a 4 percent annual reduction between 1971 and 1984, and Zimbabwe a 6.9 percent annual reduction between 1979 and 1984. For higher rates, one has to look to Asia, where Singapore, for instance, achieved a 9.1 percent annual reduction in contraceptive nonuse between 1970 and 1977.

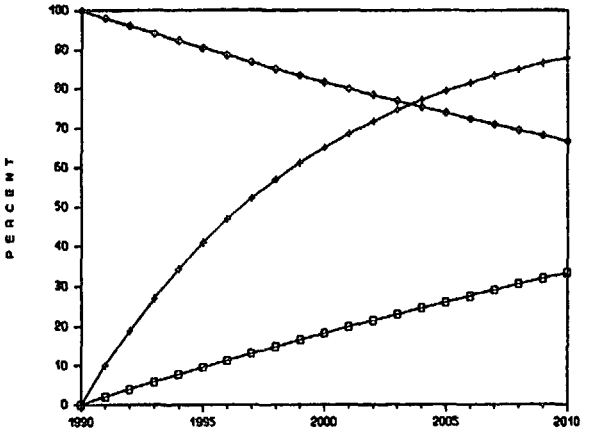
In the low-condom-use scenario, usage goes from 0 percent of all sexually active pairings in 1990 to 33 percent in 2010. In the high-condom-use scenario, usage reaches 88 percent in 2010. Figure 2 also shows an alternative scenario where number of sexual partners changes, with the average sexually active individual having only two-thirds the number of partners in 2010 as in 1990.

Figures 3 and 4 focus on the percent of adult females who are HIV-seropositive. Among adult males, the percentages are similar but not identical. These curves lag slightly behind curves for proportions seroconverting, which are very roughly a tenth as high.

Figure 3 indicates that high condom use could have a substantial effect, reducing peak seroprevalence from 13 to 8 percent and hastening the inevitable decline from peak seroprevalence by about seven years. Low condom use has a smaller effect. The figures also illustrate the effects of targeting. In the "balanced" condom use scenario, the high-sexually-active group is assumed to adopt high condom use and the low-sexually-active group low condom use, whereas in the "skewed" condom use scenario, the two groups' situations are reversed. The balanced scenario involves only 50 to 65 percent as much condom use as the skewed scenario, because the low-sexually-active group is much bigger than the high-sexually-active group. However, the balanced scenario produces much greater reduction in HIV spread.

The more modest declines assumed in numbers of sexual partners also appear to have some effect (Figure 4). Again, targeting makes a big difference. If the 2 percent annual declines

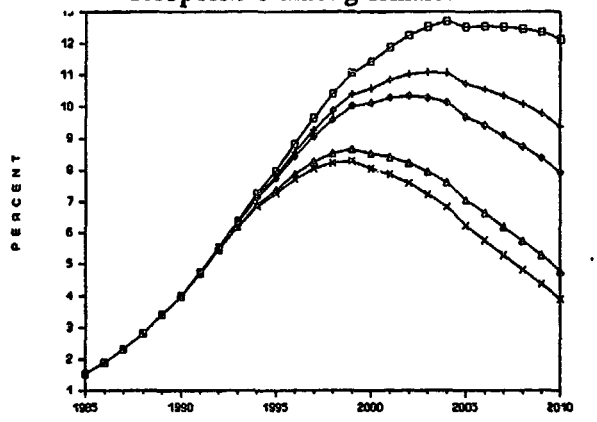
Figure 2 Assumed trends in sexual behavior



Legend

- ■ Low condom use: Percent using condoms, given that percent not using declines by 2% annually from 100% in 1990
- \+ High condom use: Percent using condoms, given that percent not using declines by 10% annually from 100% in 1990
- ♦ High sex partners: Index for number of new sex partners per year (1990=100%), with a 2% annual decline

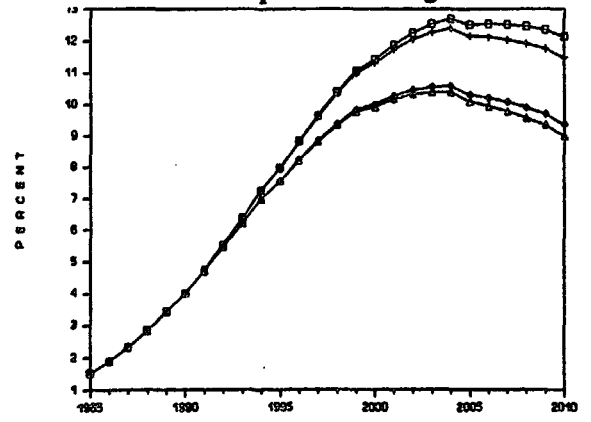
Figure 3 Effects of condom use: Percent HIV seropositive among females



Legend

- ■ Base: No condom use
- \+ Low condom use: Proportion not using condoms decreases by 2% annually from 100% in 1990
- ♦ Skewed condom use: Proportion not using condoms decreases by 10%, 5%, and 2% annually among the low, medium, and high sexually active
- ▲ Balanced condom use: Proportion not using condoms decreases by 2%, 5%, and 10% annually among the low, medium, and high sexually active
- x High condom use: Proportion not using condoms decreases by 10% annually from 100% in 1990

Figure 4 Effects of fewer sexual partners: Percent HIV seropositive among females



Legend

- ■ Base: No change in sexual partners
- \+ Skewed reduction: Number of new sexual partners decreases annually from 1990 by 2%, 1%, and 0% among the low, medium, and high sexually active
- ♦ Balanced reduction: Number of new sexual partners decreases annually from 1990 by 0%, 1%, and 2% among the low, medium, and high sexually active
- ▲ High reduction: Number of new sexual partners decreases annually from 1990 by 2%

take place mainly among the more sexually active, the outcome is almost as good as if the declines come across the board.

Effects of medical interventions

The other scenarios, collectively referred to here as "medical intervention" scenarios, are illustrated in Figure 5. The proportion with genital ulcers, assumed to be 5 percent among the most sexually active women (and much lower in all other groups), is assumed to decline to half of a percent in 1993. The proportion of injections that use sterilized needles is assumed to start at 20 percent in 1990 and to reach 60 percent in 1991 and 95 percent in 1994. And the proportion of transfusions involving screened blood is assumed to start at 10 percent in 1990 and to reach 55 percent in 1991 and 94 percent in 1994.

These changes are more extreme, in percentage terms, than the assumed changes in sexual behavior, but, as Figure 6 shows, the results on HIV transmission are negligible. None of these changes in proximate behavior appears to produce a substantial effect. Each change will be considered further.

The simulation assumed that the proportions of women with genital ulcers were 5 percent, 1 percent, and 0.1 percent in the high-sexually-active, medium-sexually-active, and low-sexually-active groups, and 0.1 percent among men in all sexual activity groups. It also assumed that the risk of transmission, given genital ulcers, was five times as great as the risk without ulcers. No basis exists for substantially raising the proportions with genital ulcers. However, the effect of ulcers on transmission is essentially unknown and might conceivably be much greater. If ulcers multiplied the risk fifty-fold rather than five-fold, the effects of reducing ulcers would be noticeable (Figure 7), though still modest relative to the effects of condom use.

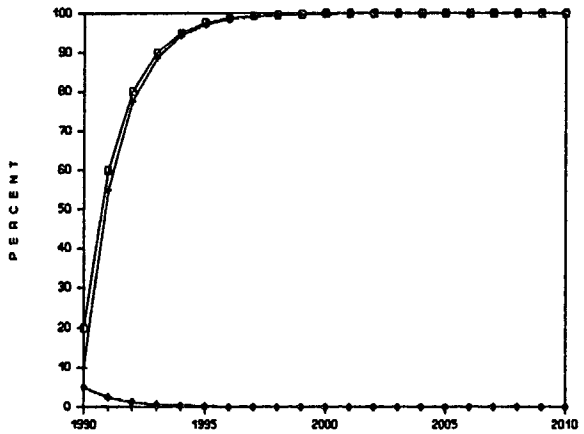
The lower lines in Figure 8 show the proportion of seroconversions due to infected injections each year--only 1 to 2 percent, in the base scenario. (Note that infections of children are not considered.) That sterilizing needles has little effect on adult infections overall is therefore not surprising. In an alternative scenario where injections were responsible for more seroconversions (because of 20 percent more injections in the population and roughly double the previously used transmission rates due to injections), the decline in seroconversions due to injections would be more noticeable, as the upper lines in Figure 8 show. However, the overall change in seroprevalence would still be quite small.

The seroconversions due to transfusions are even fewer in this simulation, ranging from 0.03 to 0.06 percent of seroconversions among adults. The effective rate for transfusions in the simulation is 0.12 per thousand. If a higher transfusion rate of 4 per thousand were used (equivalent to about half the reported rate of 630 per million per month in an urban population), the seroconversions due to transfusions would still be proportionally quite small. One cannot therefore except blood screening to have much effect, unless infection through transfusions is a much more important factor among children.

Effects of timing and targeting

The base scenario assumes that 1985 HIV seroprevalence, across the entire society, is about 1.25 percent. What if seroprevalence is lower? One way to look at this is to assume that such a country is at an earlier stage of the epidemic, but is following the same basic path. Then the same base scenario might be used, but with control measures being introduced several years earlier. Figure 9 illustrates the situation where a 2 percent decrease in proportion not using condoms starts in 1990, in 1985, or in 1980. An earlier start clearly allows the epidemic to be

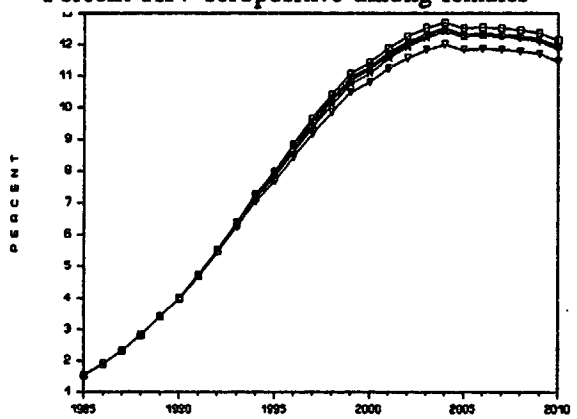
Figure 5 Assumed trends in medical interventions



Legend

- ■ Safe injections: Percentage of needles sterilized, assuming 50% annual increase from initial 20% level
- \+ Safe transfusions: Percentage of transfusions using blood or products screened for HIV, assuming 50% annual increase from initial 10% level
- ♦ Genital ulcers: Percentage of highly sexually active women with genital ulcers, assuming 50% annual decline from initial 5% level

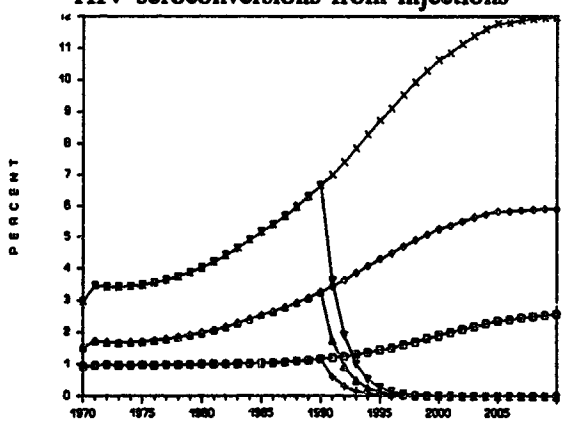
Figure 6 Effects of medical interventions: Percent HIV seropositive among females



Legend

- ■ Base: No interventions
- \+ Safe injections: Unsafe injections reduced 50% annually from 1990
- ♦ Safe transfusions: Unsafe blood supplies reduced 50% annually from 1990
- ▲ Genital ulcers: Genital ulcers reduced 50% annually from 1990
- x All interventions: Unsafe injections, unsafe blood supplies, and genital ulcers all reduced 50% annually from 1990

Figure 7 Effects of safer injections: Percent HIV seroconversions from injections



Legend

- ■ Base: No reduction in unsafe injections
- \+ Reduction: Unsafe injections reduced 50% annually
- ♦ Serious: Unsafe injections, about twice as likely to transmit HIV, not reduced
- ▲ Serious-reduction: Combination of serious and reduction
- x Severe: Unsafe injections, about twice as likely to transmit HIV and twice as frequent in population, not reduced
- ▼ Severe-reduction: Combination of severe and reduction

contained at a lower level.

Of course, currently lower HIV prevalence levels may not reflect a later start of the epidemic but rather a different configuration of transmission factors. A systematic way to look at this is to assume that countries vary along two main dimensions, one having to do with current seroprevalence level and a second with overall level of sexual activity. Under what combination of these factors might control measures be most effective?

This is investigated by looking at the effects of a 2 percent annual decrease in condom nonuse in four types of societies: high seroprevalence-high sexual activity; medium seroprevalence-high sexual activity; medium seroprevalence-low sexual activity; and low seroprevalence-low sexual activity. In comparison with the base scenario, the high-sexual-activity society is assumed to have a 10 percent greater proportion of the population in the high-sexually-active group, which also has 10 percent more new sexual partners per year. The low-sexual-activity society is defined as being 10 percent below the base scenario in both respects. Medium seroprevalence is defined as equivalent to 1985 seroprevalence in the base scenario (so that the second and third societies are matched in this respect), whereas high seroprevalence and low seroprevalence are simply the result of applying the higher or lower sexual activity rates from 1970.

Figure 10 compares the effects of condom use in the high prevalence-high sexual activity and medium prevalence-high sexual activity societies. Equivalent levels of condom use, both starting in 1990, would produce essentially the same reduction in seroprevalence among females for the period represented. A parallel comparison between low-sexual-activity societies also shows essentially no difference.

A comparison may also be made between the two medium prevalence societies, one with high and one with low sexual activity. Figure 11 shows that, in this comparison too, condom use has equivalent effects. In fact, if the comparison is between high prevalence-high sexual activity and low prevalence-low sexual activity, the effect of condom use in reducing seroprevalence still seems to be roughly equivalent.

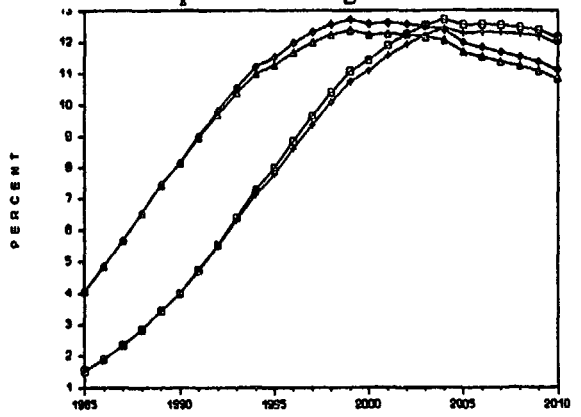
Equivalent reductions in percent seropositive imply similar AIDS case rates and death rates (assuming similar hazards), but need not translate into equivalent numbers of cases. Population sizes of the societies compared will make a difference. Nor does the same pattern of condom use--a 2 percent annual reduction in nonuse--mean exactly the same thing for different societies. In a high-sexual-activity society, it means a greater volume of condom use than in a low-sexual-activity society of the same population.

CONCLUSION

In a situation where HIV is relatively widely spread, changes in sexual behavior are essential to reduce the scale of the epidemic. Earlier changes are more effective than later changes. However, between countries where prevalence and sexual activity vary within specified ranges, changes in sexual behavior do not appear likely to produce widely different effects.

The other proximate determinants do not appear as important for overall control of HIV transmission. However, they may be important for other reasons: to guarantee the integrity of the health delivery system, for instance, or as part of a broad-scale attack on the disease in which synergisms among control measures may be critical. As Figure 1 illustrates, a program to control sexually transmitted diseases may have not only direct effects on genital ulcers but also effects in promoting condoms and reducing numbers of sexual partners. Given the vast uncertainties about how to change sexual behavior, such linkages should not be ignored.

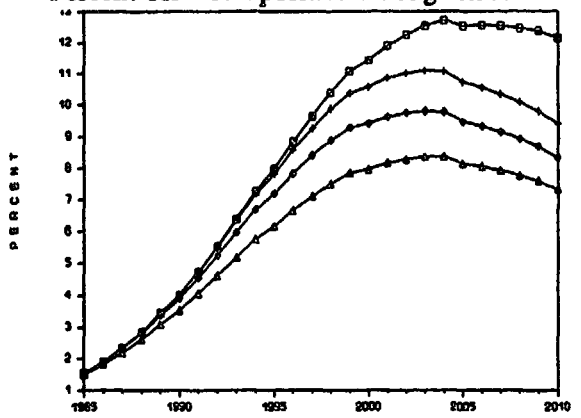
Figure 8 Effects of STD control: Percent HIV seropositive among females



Legend

- ■ Base: Genital ulcers raise susceptibility five-fold, do not decrease in prevalence
- \+ High STD control: Genital ulcers raise susceptibility five-fold, reduced in prevalence 50% annually
- ♦ Severe: Genital ulcers raise susceptibility fifty-fold, do not decrease in prevalence
- ▲ Severe-high control: Genital ulcers raise susceptibility fifty-fold, reduced in prevalence 50% annually

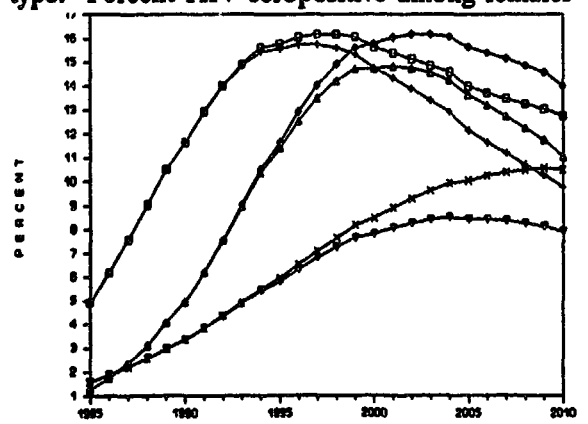
Figure 9 Effects of timing of condom program: Percent HIV seropositive among females



Legend

- ■ Base: No condom use
- \+ 1990 start for 2% annual decrease in condom nonuse
- ♦ 1985 start for 2% annual decrease in condom nonuse
- ▲ 1980 start for 2% annual decrease in condom nonuse

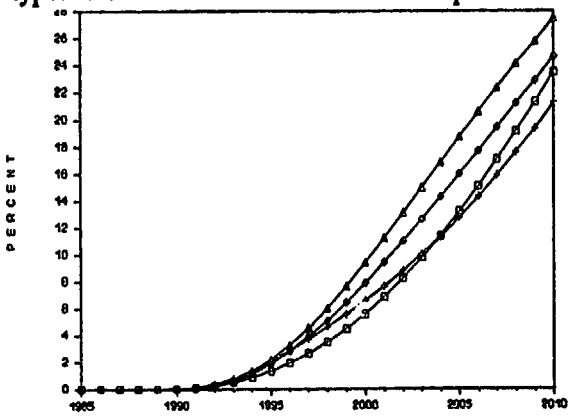
Figure 10 Effects of condom use by country type: Percent HIV seropositive among females



Legend

- ■ High 1985 HIV seroprevalence, relatively high sexual activity, no condom use
- \+ High 1985 HIV seroprevalence, relatively high sexual activity, condom use increasing
- ♦ Medium 1985 HIV seroprevalence, relatively high sexual activity, no condom use
- ▲ Medium 1985 HIV seroprevalence, relatively high sexual activity, condom use increasing
- x Medium 1985 HIV seroprevalence, relatively low sexual activity, no condom use
- ▼ Medium 1985 HIV seroprevalence, relatively low sexual activity, condom use increasing

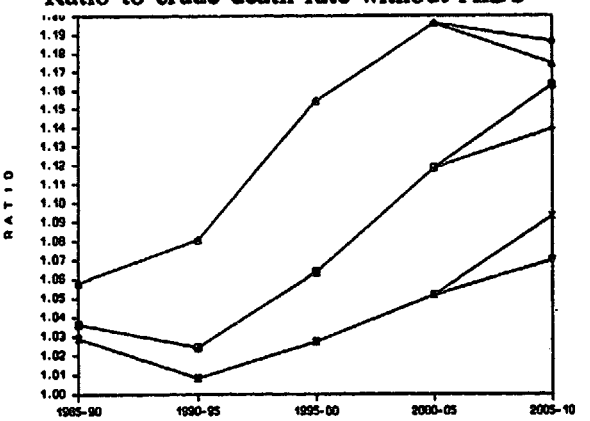
Figure 11 Effects of condom use by country type: Percent reduction in HIV seropositives



Legend

- ■ High 1985 HIV seroprevalence, relatively high sexual activity
- \+ Medium 1985 HIV seroprevalence, relatively high sexual activity
- ♦ Medium 1985 HIV seroprevalence, relatively low sexual activity
- ▲ Low 1985 HIV seroprevalence, relatively low sexual activity

Figure 12 Country type and crude death rates: Ratio to crude death rate without AIDS



Legend

- ■ Medium 1985 HIV seroprevalence, medium sexual activity, no condom use
- \+ Medium 1985 HIV seroprevalence, medium sexual activity, condom use increasing
- ♦ High 1985 HIV seroprevalence, relatively high sexual activity, no condom use
- ▲ High 1985 HIV seroprevalence, relatively high sexual activity, condom use increasing
- x Low 1985 HIV seroprevalence, relatively low sexual activity, no condom use
- ▼ Low 1985 HIV seroprevalence, relatively low sexual activity, condom use increasing

PROJECTING THE DEMOGRAPHIC IMPACT OF THE HIV EPIDEMIC USING STANDARD PARAMETERS

Rodolfo A. Bulatao

A model for estimating the demographic impact of the spread of the Human Immunodeficiency Virus (HIV) is described, and model results are presented using standard parameters chosen with reference to the situation in the African acquired immunodeficiency syndrome (AIDS) belt. Alternative scenarios are presented but discussed only briefly.

STRUCTURE OF THE MODEL

The model has two parts: an epidemiological model that provides a numerical microsimulation of the spread of HIV and a demographic model that translates tbe epidemiological results into population outcomes. The demographic model can be covered briefly and will be considered first. The epidemiological model is more complicated and requires some elaboration.

Demographic model

The demographic model is a standard cohort-component model that, given an initial age-sex structure and a sequence of vital rates, provides an array of outputs (partly illustrated in Table 3) for periods up to 75 years. The population and vital rate data to be used as input are not limited: they can be generated by any appropriate algorithm or they can even be fluctuating or discontinuous. The model can be and has been used independently of the epidemiological model to generate population projections.

The model projects population by quinquennium rather than annually, linking with the epidemiological model, which makes annual estimates, every five years. Annual estimates from the epidemiological model may therefore be discontinuous around the points of linkage.

One specific limitation of the demographic model is that it does not deal with marriage. Linked with the epidemiological model, the demographic model also loses some capabilities it otherwise has. First, it cannot be used for urban-rural projections, although the epidemiological model by itself can generate some urban-rural outcomes. Second, it cannot be used for backward projection. Third, requests for special output, such as information on special age groups, cannot be filled (though they could be filled by an independent run of the demographic model).

Epidemiological model

The epidemiological model allows HIV infection to spread sexually between partners; through blood and needles; and vertically from mother to child. Each mode of transmission is discussed separately below. Progression from infection to AIDS, and then to death, which is independent of transmission mode, is then discussed. Next, complications to the basic model meant to allow for testing the effects of a variety of parameters are described.

Sexual transmission. In handling the sexual transmission of HIV, the model first divides all adults (15 years old and over) into four sexual orientation groups: females (assumed to be all heterosexual), heterosexual males, homosexual males, and bisexual males. Females are assumed to choose partners from among heterosexual and bisexual males; heterosexual males from among

females; homosexual males from among themselves and bisexual males; and bisexual males from females, homosexual males, and among themselves. Within each group, up to ten subgroups varying by sexual behavior can be defined. Those who have developed AIDS are assumed not to be sexually active, and therefore are effectively in a no-sex subgroup.

Given some initial assumption about the proportion infected with HIV in a particular year, the number sexually infected in each subsequent year is calculated subgroup by subgroup. In its basic form, the calculation of the number of HIV seroconversions (O) in one year (t) in a given subgroup (s) of one sexual orientation group (r) is given by

$$O_{rst} = M_{rs,t-1} P_{t-1} \{1 - (1 - O_I) (1 - O_T) (1 - R_{r,t-1} [1 - (1 - T_r)^{Sr}])^{Frs}\} \quad (1)$$

where M is the number of HIV seronegatives in the subgroup, P is the likelihood of adult survival from mortality unrelated to HIV, R is the proportion of potential sexual contacts, or agents, who are infective, T is the likelihood of transmission to group r through sexual contact, S is an average number for sex acts per partner, and F is the annual number of new partners. (Different values of F and S are the main factors distinguishing sexual orientation subgroups.) O_I and O_T will be explained below.

The proportion of agents (R) infective to subgroup s is the weighted proportion across all other subgroups with which subgroup s has sexual contact who are asymptomatic but HIV-seropositive (A). The weights used are the frequencies (F) with which these other subgroups take new sexual partners. The asymptomatic seropositives (A) in a given subgroup are calculated by summing up all those infected in previous years and subtracting those expected to progress to AIDS or to die from other causes.

No restrictions are placed on the way sexual orientation groups are divided into subgroups or on the values of F and S specified for each subgroup. Inconsistent parameters, requiring for example that males have many more sexual partners in the aggregate than the available females, can be used as input. The model adjusts the input figures if necessary, essentially taking averages or adjusting figures proportionally, in order to force them into consistency. These adjustments are made for each year of the simulation, as the number and distribution of the sexually active changes.

Nonsexual transmission. Infection with HIV through needles and blood accounts for the terms O_I and O_T in equation (1). The adult population is divided into crosscutting subgroups for number of annual tranfusions and number of annual injections, with the bulk of adults typically assumed to be in zero-frequency subgroups. The probability of infection (O_I or O_T) in each subgroup is then a function of the proportion of "agents" (needles or blood) infected, the likelihood of transmission through a single exposure to an infected agent, and the frequency of such exposure, through injections or transfusions, for the particular subgroup. The formula is similar to (1), leaving out a few irrelevant terms.

The proportion of needles infected is equal to the proportion of adults infected, weighted by the frequency with which each receives an injection. The proportion of blood infected is equal to the proportion of adults infected, optionally weighted by propensity to donate blood, which can be specified for each sexual orientation subgroup. Both these proportions can be modified, the first by a specified proportion of needles sterilized and the second by a specified proportion of blood screened.

For vertical transmission, the number of infected newborns is the product of the number of women of reproductive age, the proportion of women infected (including, in this case,

those who have progressed to AIDS), the general fertility rate, and an optional adjustment factor that can be used if it is assumed that those infected have either higher or lower fertility than others. The model does not allow overall fertility to be affected by use of this adjustment factor, implying in effect that fertility among those not infected either rises or falls to counterbalance the adjustment. Nor does the model make allowance for children being infected in any other fashion, nor for their transmitting the virus to anyone else.

Progression. The number that progress to AIDS is determined by applying separate schedules for adults and children that depend on year since initial infection, after excluding mortality from other causes. The model therefore keeps track of cohorts of HIV seropositives distinguished by infection date, so that appropriate progression rates can be applied. A logistic function is presently applied in generating the schedule, but alternative functions can in principle be incorporated. Progression from AIDS to death due to AIDS is handled in a similar manner.

The model keeps track of age at death due to AIDS among children. Deaths due to AIDS among adults are distributed across five-year age groups according to an age-sex distribution provided by the user. These are translated into probabilities of dying, which, because they already exclude mortality from other causes, can be simply added to the life table values that are among the vital rates that drive the demographic model. Some aggregation is necessary because the epidemiological model generates annual estimates, while the demographic model generates quinquennial estimates.

Model complications. Several complications of the model mainly affect the transmission process and allow tests of the effects of various unknown parameters. These complications can be ignored in a routine run of the model.

Random movement between sexual orientation subgroups. Membership in the four main sexual orientation groups is assumed to be fixed. Within these groups, subgroup proportions are initially specified by the user, and these proportions are applied to the initial population and to all those entering adulthood each year. In one scenario, each individual stays in the same subgroup all his or her life. In an alternative scenario, some proportion of individuals moves between subgroups each year. The model allows specification of a fixed proportion of individuals who move randomly among subgroups, with the likelihood that they enter a given subgroup being proportional to the distribution across subgroups. This is accomplished computationally by redistributing the specified proportion of individuals each year before calculating seroconversions.

Infective phases. Different probabilities of transmission can be entered for contact between susceptibles and particular infective agents--men, women, needles, blood, mothers. In addition, human agents can be allowed to vary in infectivity over time. The period between infection and development of AIDS can be divided into up to six segments of varying length, for each of which a specific probability of transmission can be specified. (Since the entire period varies in length across individuals, the segments are proportional rather than of absolute length.) Computationally, this affects equation (1), in which the term

$$R_{r,t-1}\,[1 - (1 - T_r)^{S_r}]$$

is actually calculated as the summation of similar terms for each of the infective phases.

Cofactors. The model allows condom use, genital ulcers, and one user-specified cofactor to affect infection by acting as a multiplier of transmission rates. In the case of condoms, for instance, transmission rates may be multiplied by .1 or .2 or some other value for those using condoms. An appropriate multiplier and the proportion in each sexual orientation subgroup to

whom the cofactor applies must be specified. From period to period, those to whom the cofactor applies may stay essentially the same or may change randomly.

Anal sex. On the assumption that anal sex carries different risks from other forms of sex, the user is allowed to specify what this greater risk is and what proportion of sex acts involve anal intercourse for each sexual orientation subgroup.

Genetic variability. The adult population can be divided into two or three groups of arbitrary size with varying susceptibilities to infection. These groups crosscut the sexual orientation groups. A multiplier for the transmission rates and a relative size must be specified for each group. Computationally, this creates additional compartments in the model but does not change the basic formula.

Changes in parameters. The model allows changes over time in several parameters. This is accomplished by selecting a date for changes to start and a percentage by which the parameter will rise or fall annually. Changes are allowed in all these parameters:

transmission probabilities through sex, needles, and blood
perinatal transmission probabilities
proportion of needles sterilized
proportion of blood screened
number of sex partners for each subgroup
number of sex acts per partner
proportion using condoms
proportion with genital ulcers
proportion with a third user-specified cofactor
proportion of sex acts involving anal intercourse

For condom use--a factor of particular interest--change is specified as the annual decrease in the proportion of sex acts in which condoms are *not* used. This specification reflects the greater difficulty of increasing contraceptive use once high levels have been reached, and also ensures that condom use will never exceed 100 percent.

Attraction matrix. The basic model assumes proportionate mixing among the relevant subgroups, modified by the frequency with which each subgroup chooses partners. A further modification can be made if one wishes to assume that contact between particular subgroups is more likely than contact between others. An "attraction matrix" is defined providing weights for the likelihood of contact between every pair of subgroups. These weights are used in calculating the proportion of agents (R) that are infective to a given subgroup. A weight of 2, for instance, implies in effect that members of each of the two subgroups, in looking over possible partners, behave as if there were twice as many members of the other subgroup as there actually are. As with other sexual behavior parameters, the attraction matrix can be input in an unbalanced form, and will be forced into balance, which is accomplished through an iterative process.

Urban-rural differences. By defining additional subgroups and using an attraction matrix, it is possible to run a simulation with separate urban and rural sectors. Each sexual orientation subgroup must be divided into two groups, one rural and one urban. The attraction matrix can then be used to increase likelihood of contact within sectors and reduce contact across sectors. In addition, if the proportion of the population in the urban sector in each year is defined, the model shifts sufficient numbers of people between the parallel subgroups in each sector to meet these figures.

Outputs. To the output of the demographic model the epidemiological model adds information on HIV infections and outcomes. For each sexual orientation group, as well as for all adults and all children, the epidemiological model provides annual estimates of seronegatives, seroconversions, asymptomatic seropositives (classified by infective phases, if these are introduced), AIDS incidence, AIDS prevalence, and AIDS deaths. For convenience, the output also includes cumulative numbers of AIDS cases and deaths for children, adult males, and adult females, seroconversion rates for adults by sex, and proportions seropositive by sexual orientation group.

Estimates of HIV infection by sexual orientation subgroup are not routinely printed but can be extracted from the model. This is not the case for subgroup estimates of AIDS incidence, which are not calculated.

APPLICATION

The model was run with the standard parameters, with some minor adjustments and additional assumptions. The adjustments and assumptions will be discussed first. Because the maximum and minimum values of the standard parameters gave very divergent results, a medium variant using intermediate parameters was also run. The results for these high, low, and medium variants will be presented. Finally, consideration will be given to alternative projections varying other parameters unspecified in the standard variants.

Adjustments and assumptions

The standard parameters include specification of a hypothetical country's demographic situation, the epidemiology of HIV, and the sexual behavior of the population. In most cases, these parameters were used as input essentially as recommended. The cases where this was not so will be noted. Then the specific parameters distinguishing high, medium, and low variants will be summarized.

Demographic parameters. The demographic parameters presented no problems. The model could not exactly duplicate the standard scenario, but it did come quite close. For instance, the recommended survivorship ratios gave a female life expectancy in the last quinquennium of 62.9 (before the effect of HIV is introduced), slightly below the recommended value of 63. Similarly, the recommended age-specific fertility rates do not add up exactly to the recommended total fertility rate; in this case, the total was maintained and the age-specific rates adjusted slightly.

Epidemiological parameters. The epidemiological parameters of concern involve transmission, progression, and initial seroprevalence. The recommended values for probability of transmission were taken to apply from the point of infection up to the point when an individual is eliminated from the pool of infectives. The rate was assumed to be constant over this period. Since the model does not allow children to be infected other than vertically, infections of children through transfusions were not represented.

For adult progression to AIDS, the recommended function is linear. This was approximated fairly closely in the model runs with a logistic function, as was the progression from AIDS to death (Figures 1 and 2). For children, the model runs assumed that 50 percent of those infected developed AIDS by the first year, 98 percent by the second year. The model runs also assumed that, one year after developing AIDS, 50 percent had died; two years after, 88 percent had died; and three years after, 98 percent had died.

The recommended value for initial seroprevalence (in 1985) was 3.43 percent. To obtain an appropriate distibution of those infected across sexual orientation subgroups, it is useful

Figure 1 Cumulative percentage of adults with AIDS, by year since infection: Standard and model estimates

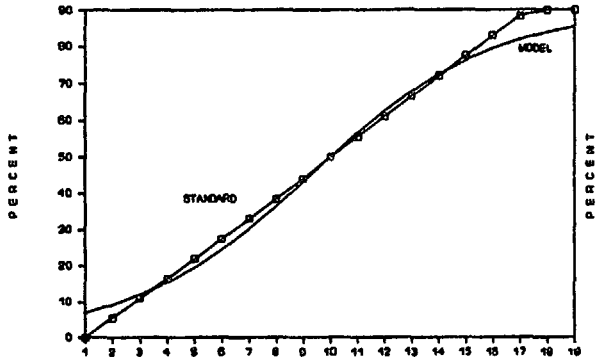
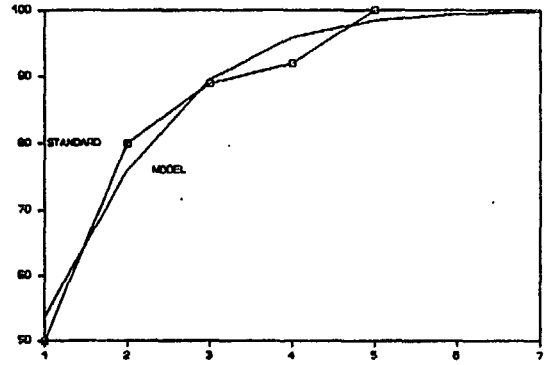


Figure 2 Cumulative percentage of adults dead, by year since developing AIDS: Standard and model estimates



to start the model several years before the initial year, allowing the model to generate an appropriate 1985 distribution. The model was therefore run from 1980, with starting seroprevalence values chosen by experiment to give the desired 1985 seroprevalence. (An earlier start is desirable but was not possible because the high and low variants gave such extreme results.)

Sexual behavior. The standard parameters include the requirement that those with full-blown AIDS continue to be sexually active. This is not allowed in the model and was therefore not done. On the other hand, the model requires at least some homosexuals and bisexuals. Together, the two groups were set at 1 per 100,000 of the adult population.

The parameters also include distributions of the population by sexual orientation. Table 1 shows how these were translated into model parameters. Single adults were estimated given the standard marriage rates. It was assumed that all prostitutes are single. In addition, monogamous married individuals were divided for convenience into those with monogamous and promiscuous spouses. The annual numbers of partners for promiscuous women and prostitutes could then be estimated, and are shown below in Table 2.

The model requires specification of number of sex acts per partner, but allows only one figure per sexual orientation subgroup. This poses a problem for subgroups composed of promiscuous married individuals, who, according to the standard parameters, have multiple contacts with a spouse but only one contact each with a variety of other partners. Rough estimates of appropriate figures were made, and are also given below in Table 2.

High, medium, and low variants. Table 2 summarizes the parameters that differ between these variant runs of the model. The parameters for the medium variant are close to the averages for the high and low variants.

Table 1 Assumed distribution of adult population by marital status and sexual activity

Marital status and sexual activity	Percent Males	Females
Single	13.0	
Nonprostitute		9.7
Prostitute		4.3
Married		
Monogamous		
With monogamous spouse	15.0	15.0
With promiscuous spouse	11.0	35.0
Promiscuous		36.0
With nonprostitutes	35.0	
With prostitutes	26.0	
Total	100.0	100.0

Table 2 Parameters that differ between low, medium, and high variants

Parameter	Low	Medium	High
Transmission			
Female to male	0.001	0.01	0.1
Male to female	0.003	0.03	0.1
Percent of sex acts involving condoms	2	10	20
Annual partners, excluding any spouse			
Male promiscuous with nonprostitutes	4	8	12
Males promiscuous with prostitutes	12	24	36
Female prostitutes	71	143	214
Other promiscuous females	6.3	12.6	19
Sex acts per partner			
Male promiscuous with nonprostitutes	10.0	4.3	1.3
Males promiscuous with prostitutes	4.3	2.1	1.1
Other promiscuous females	10.0	2.8	1.3
Percent of adults seropositive, 1985	3.39	3.41	3.38

Results

Table 3 first presents a baseline demographic projection, in which HIV infection is assumed to have no demographic effect. As already noted, this matches the standard parameters quite closely. Values of e_{20} are not normally generated, but have been calculated and added to the table.

Tables 4 to 9 then present the low, medium, and high variants for the effect of HIV infection. The first of each of these pairs of tables shows estimated cases of HIV infection and of AIDS, as well as incidence and prevalence rates when these are calculated by the model. Estimates are given by single year rather than by quinquennium, because the model calculates them this way (because year-to-year variation can be substantial). Adults (15 years old and over) are distinguished from children, but smaller age groups are not distinguished, as noted above, until deaths take place.

Each variant starts with a slightly different number of cumulative cases in 1985. This is necessary to equalize initial HIV seroprevalence levels: given the differences in parameters among variants, a different time pattern of infections is needed to achieve the same 1985 prevalence.

The second table in each pair (i.e., Tables 5, 7, and 9) then presents the demographic projection linked to the epidemiological results. These demographic results are presented by quinquennium and for five-year age groups.

Table 10, 11, and 12, finally, present numbers of new cases of HIV infection for the low, medium, and high variants. These statistics are not normally output by the program but can be requested.

The great divergence between the low and the high variants should be noted. (To highlight the differences, Figure 3 shows female seroprevalence levels, and Figure 4 shows life expectancies for both sexes combined.) In the low variant, the proportion infected never rises above the starting value. In the high variant, as much as 60 percent of the adult population becomes infected. The contrast is largely due to the thirty-fold to one-hundred-fold difference between the variants in transmission rates from sexual contact. In separate simulations with the transmission rates alone interchanged, the results are reversed, with the low variant showing over 60 percent prevalence and the high variant showing almost no infection.

Figure 3 Adult female seroprevalence: high, medium, and low variants, by year

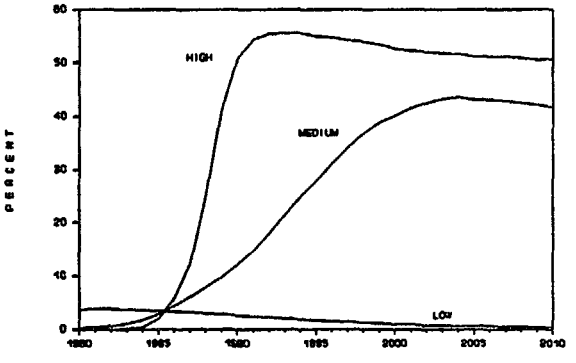


Figure 4 Projected life expectancy: high, low, and medium variants, by quinquennium

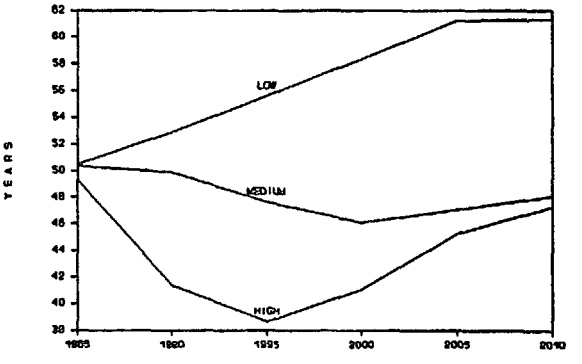


Table 3 Population projection without AIDS

AGE GROUP	1985	1990	1995	2000	2005	2010
TOTAL M+F	200000	238372	282095	331899	388468	452133
MALES TOTAL	100000	119163	141021	165943	194278	226179
0-4	20102	24012	27111	30514	34233	38083
5-9	15846	18823	22686	25847	29351	33218
10-14	12970	15482	18442	22290	25467	29000
15-19	10679	12716	15208	18153	21984	25167
20-24	8726	10398	12410	14879	17801	21609
25-29	7088	8447	10095	12084	14530	17434
30-34	5750	6849	8188	9815	11785	14213
35-39	4649	5537	6618	7938	9548	11503
40-44	3734	4449	5319	6383	7686	9281
45-49	2972	3539	4236	5089	6134	7420
50-54	2330	2776	3324	3999	4828	5850
55-59	1789	2130	2554	3077	3725	4526
60-64	1327	1581	1898	2294	2785	3397
65-69	930	1108	1334	1618	1975	2421
70-74	593	708	858	1050	1294	1605
75+	515	607	738	914	1146	1450
FEMALES TOTAL	100000	119209	141074	165957	194195	225953
0-4	19791	23695	26703	30008	33615	37341
5-9	15642	18582	22442	25510	28909	32653
10-14	12811	15291	18218	22067	25156	28590
15-19	10557	12574	15040	17956	21797	24901
20-24	8671	10330	12331	14783	17690	21522
25-29	7095	8452	10095	12082	14521	17421
30-34	5782	6889	8231	9861	11836	14268
35-39	4692	5589	6682	8012	9631	11600
40-44	3787	4513	5396	6475	7792	9401
45-49	3042	3623	4334	5202	6266	7568
50-54	2422	2884	3449	4142	4992	6037
55-59	1896	2257	2701	3247	3918	4745
60-64	1440	1715	2056	2477	2996	3640
65-69	1037	1236	1486	1798	2186	2668
70-74	684	817	987	1202	1473	1814
75+	649	762	922	1136	1417	1782
BIRTH RATE		49.9	46.7	43.9	41.3	38.8
DEATH RATE		14.8	13.1	11.4	9.9	8.4
RATE OF NAT. INC.		3.51	3.37	3.25	3.15	3.04
NET MIGRATION RATE		.0	.0	.0	.0	.0
GROWTH RATE		3.51	3.37	3.25	3.15	3.04
TOTAL FERTILITY		7.300	6.800	6.300	5.800	5.300
NRR		2.679	2.582	2.475	2.357	2.227
e(0) - BOTH SEXES		51.46	53.75	56.17	58.72	61.40
e(10) - BOTH SEXES		53.23	54.46	55.74	57.09	58.49
e(20) - BOTH SEXES		45.39	46.14	47.23	48.38	49.59
IMR - BOTH SEXES		101.2	91.2	81.2	71.2	61.3
q(5) - BOTH SEXES		.1661	.1480	.1296	.1111	.0924
DEP. RATIO	103.2	103.3	101.3	97.6	92.1	87.2

Table 4 HIV and AIDS cases, for adults and children, by year: Low variant

	HIV incidence (percent)		HIV prevalence (per thousand)		Cumulative HIV cases (thousands)			Current AIDS cases (thousands)			Cumulative AIDS cases (thousands)		
Year	Adult female	Adult male	Adult female	Adult male	Adult female	Adult male	Children	Adult female	Adult male	Children	Adult female	Adult male	Children
1985	2.4	1.9	3.4	3.3	0.8	0.7	0.8	0.1	0.1	0.2	0.3	0.3	0.6
1986	2.3	1.8	3.3	3.2	0.9	0.8	1.0	0.1	0.1	0.2	0.4	0.4	0.8
1987	2.1	1.6	3.2	3.0	1.0	0.9	1.1	0.2	0.2	0.2	0.5	0.5	0.9
1988	1.9	1.5	3.0	2.8	1.1	1.0	1.3	0.2	0.2	0.2	0.6	0.6	1.1
1989	1.8	1.3	2.8	2.6	1.2	1.0	1.4	0.2	0.2	0.2	0.8	0.7	1.2
1990	1.6	1.2	2.6	2.3	1.3	1.1	1.6	0.2	0.2	0.2	0.9	0.8	1.3
1991	1.4	1.1	2.4	2.1	1.4	1.2	1.7	0.2	0.2	0.2	1.0	0.9	1.5
1992	1.3	1.0	2.2	1.9	1.5	1.2	1.9	0.2	0.2	0.2	1.1	1.1	1.6
1993	1.2	0.9	2.0	1.7	1.6	1.3	2.0	0.2	0.2	0.2	1.2	1.1	1.7
1994	1.1	0.8	1.8	1.6	1.6	1.3	2.1	0.2	0.2	0.2	1.3	1.2	1.8
1995	0.9	0.7	1.6	1.4	1.7	1.4	2.2	0.2	0.2	0.2	1.4	1.3	1.9
1996	0.9	0.6	1.5	1.2	1.8	1.4	2.3	0.2	0.1	0.2	1.5	1.4	2.0
1997	0.8	0.6	1.4	1.1	1.8	1.5	2.4	0.2	0.1	0.1	1.6	1.5	2.1
1998	0.7	0.5	1.3	1.0	1.9	1.5	2.5	0.1	0.1	0.1	1.7	1.5	2.1
1999	0.7	0.5	1.2	1.0	2.0	1.6	2.5	0.1	0.1	0.1	1.7	1.6	2.2
2000	0.6	0.4	0.9	0.7	2.0	1.6	2.6	0.1	0.1	0.1	1.8	1.6	2.3
2001	0.5	0.4	0.8	0.6	2.0	1.6	2.7	0.1	0.1	0.1	1.8	1.7	2.4
2002	0.4	0.3	0.8	0.6	2.1	1.7	2.7	0.1	0.1	0.1	1.9	1.7	2.4
2003	0.4	0.3	0.7	0.5	2.1	1.7	2.8	0.1	0.1	0.1	1.9	1.8	2.5
2004	0.3	0.3	0.6	0.5	2.2	1.7	2.8	0.1	0.1	0.1	2.0	1.8	2.5
2005	0.3	0.2	0.5	0.4	2.2	1.8	2.9	0.1	0.1	0.1	2.0	1.8	2.5
2006	0.3	0.2	0.5	0.4	2.2	1.8	2.9	0.1	0.1	0.1	2.1	1.9	2.6
2007	0.3	0.2	0.4	0.3	2.2	1.8	2.9	0.1	0.1	0.1	2.1	1.9	2.6
2008	0.2	0.2	0.4	0.3	2.3	1.8	3.0	0.1	0.1	0.1	2.2	1.9	2.6
2009	0.2	0.2	0.4	0.3	2.3	1.8	3.0	0.1	0.1	0.1	2.2	1.9	2.7
2010	0.2	0.1	0.3	0.3	2.3	1.9	3.0	0.1	0.0	0.0	2.2	2.0	2.7

Table 5 Population projection with AIDS: Low variant.

AGE GROUP	1985	1990	1995	2000	2005	2010
TOTAL M+F	200000	237660	280692	329932	386008	449134
MALES TOTAL	100000	118807	140323	164970	193063	224708
0-4	20102	23823	26930	30342	34043	37833
5-9	15846	18748	22442	25627	29153	33013
10-14	12970	15482	18369	22050	25249	28804
15-19	10679	12716	15208	18081	21747	24953
20-24	8726	10398	12410	14878	17731	21376
25-29	7088	8441	10087	12078	14526	17362
30-34	5750	6836	8166	9796	11772	14205
35-39	4649	5524	6589	7905	9522	11486
40-44	3734	4431	5286	6340	7645	9249
45-49	2972	3522	4199	5041	6083	7374
50-54	2330	2766	3295	3954	4778	5798
55-59	1789	2125	2538	3046	3681	4476
60-64	1327	1576	1887	2275	2754	3355
65-69	930	1104	1326	1605	1957	2393
70-74	593	707	854	1043	1284	1590
75+	515	607	737	910	1139	1440
FEMALES TOTAL	100000	118853	140369	164961	192945	224425
0-4	19791	23513	26526	29838	33427	37096
5-9	15642	18504	22203	25293	28714	32450
10-14	12811	15291	18141	21831	24941	28396
15-19	10557	12574	15039	17881	21564	24689
20-24	8671	10330	12331	14783	17616	21292
25-29	7095	8441	10081	12070	14514	17343
30-34	5782	6868	8194	9826	11811	14251
35-39	4692	5569	6636	7955	9584	11566
40-44	3787	4496	5355	6414	7726	9347
45-49	3042	3613	4305	5152	6200	7500
50-54	2422	2877	3430	4107	4939	5970
55-59	1896	2253	2689	3224	3882	4693
60-64	1440	1713	2048	2463	2974	3605
65-69	1037	1235	1483	1790	2173	2648
70-74	684	817	986	1199	1467	1803
75+	649	762	922	1135	1415	1776
BIRTH RATE		50.0	46.8	44.0	41.4	38.8
DEATH RATE		15.5	13.6	11.7	10.0	8.5
RATE OF NAT. INC.		3.45	3.33	3.23	3.14	3.03
NET MIGRATION RATE		.0	.0	.0	.0	.0
GROWTH RATE		3.45	3.33	3.23	3.14	3.03
TOTAL FERTILITY		7.300	6.800	6.300	5.800	5.300
NRR		2.640	2.553	2.456	2.346	2.221
e(0) - BOTH SEXES		50.49	52.89	55.59	58.37	61.19
e(10) - BOTH SEXES		52.72	53.91	55.37	56.88	58.37
e(20) - BOTH SEXES		44.54	45.57	46.85	48.18	49.47
IMR - BOTH SEXES		105.1	93.8	82.9	72.2	61.8
q(5) - BOTH SEXES		.1754	.1547	.1340	.1137	.0939
DEP. RATIO	103.2	103.0	100.8	97.2	92.0	87.2

Table 6 HIV and AIDS cases, for adults and children, by year: Medium variant

	HIV incidence (percent)		HIV prevalence (per thousand)		Cumulative HIV cases (thousands)			Current AIDS cases (thousands)			Cumulative AIDS cases (thousands)		
Year	Adult female	Adult male	Adult female	Adult male	Adult female	Adult male	Children	Adult female	Adult male	Children	Adult female	Adult male	Children
1985	12.5	18.7	2.9	3.9	1.5	2.0	0.2	0.1	0.1	0.1	0.1	0.1	0.1
1986	16.8	28.4	4.4	6.5	2.4	3.5	0.3	0.1	0.1	0.1	0.2	0.2	0.2
1987	19.5	36.4	6.0	9.8	3.5	5.5	0.5	0.1	0.2	0.2	0.3	0.4	0.4
1988	22.0	38.9	7.8	13.0	4.7	7.7	0.8	0.2	0.3	0.3	0.4	0.6	0.6
1989	26.2	37.0	9.8	15.7	6.3	9.8	1.2	0.3	0.5	0.4	0.6	0.9	0.9
1990	31.2	33.9	12.0	17.6	8.2	11.9	1.8	0.4	0.6	0.6	0.8	1.3	1.3
1991	37.5	33.8	14.8	19.5	10.5	13.9	2.4	0.5	0.7	0.8	1.2	1.7	1.9
1992	43.3	35.0	17.9	21.4	13.4	16.2	3.2	0.7	0.9	1.0	1.6	2.3	2.5
1993	48.5	37.5	21.3	23.3	16.6	18.6	4.3	0.8	1.0	1.2	2.1	2.9	3.3
1994	52.7	40.4	24.9	25.3	20.2	21.3	5.5	1.1	1.2	1.5	2.8	3.7	4.3
1995	54.3	42.4	27.8	26.9	24.1	24.2	6.9	1.3	1.4	1.8	3.6	4.5	5.5
1996	56.3	45.3	31.1	28.9	28.3	27.5	8.6	1.6	1.7	2.1	4.6	5.5	6.9
1997	56.3	47.0	34.1	31.0	32.6	30.9	10.4	1.9	1.9	2.4	5.7	6.7	8.5
1998	55.3	47.9	36.8	32.9	36.8	34.5	12.5	2.2	2.1	2.7	7.0	7.9	10.3
1999	53.5	48.1	38.9	34.7	41.1	38.2	14.7	2.5	2.4	3.0	8.5	9.3	12.2
2000	50.8	47.0	40.2	35.7	45.2	41.9	17.1	2.8	2.6	3.3	10.2	10.8	14.4
2001	49.4	46.6	41.5	37.0	49.4	45.7	19.6	3.1	2.8	3.5	12.0	12.5	16.6
2002	47.3	45.5	42.5	38.1	53.4	49.5	22.2	3.4	3.0	3.7	13.9	14.2	18.9
2003	45.5	44.1	43.2	38.9	57.3	53.3	24.8	3.6	3.2	3.8	16.0	16.1	21.3
2004	43.9	42.7	43.6	39.4	61.2	57.0	27.5	3.9	3.4	3.9	18.3	18.0	23.8
2005	42.0	40.7	43.1	39.1	65.0	60.7	30.2	4.1	3.6	4.0	20.6	20.1	26.3
2006	41.8	40.0	43.0	39.1	68.9	64.3	32.9	4.3	3.8	4.1	23.1	22.2	28.8
2007	41.0	38.9	42.8	39.0	72.8	68.0	35.6	4.5	4.0	4.1	25.6	24.4	31.4
2008	40.4	38.0	42.5	38.8	76.7	71.7	38.3	4.7	4.1	4.1	28.2	26.7	33.9
2009	39.9	37.3	42.1	38.4	80.6	75.3	41.0	4.8	4.2	4.1	30.9	29.1	36.4
2010	39.5	36.5	41.6	37.9	84.5	79.0	43.6	4.9	4.3	4.0	33.6	31.5	38.9

Table 7 Population projection with AIDS: Medium variant

AGE GROUP	1985	1990	1995	2000	2005	2010
TOTAL M+F	200000	237587	278135	319928	362991	409394
MALES TOTAL	100000	118744	138945	159835	181513	204984
0-4	20102	23797	26273	28393	30673	33443
5-9	15846	18759	22180	24231	25926	28135
10-14	12970	15482	18379	21792	23873	25614
15-19	10679	12716	15208	18091	21493	23593
20-24	8726	10398	12410	14878	17741	21127
25-29	7088	8437	10057	12000	14393	17196
30-34	5750	6830	8105	9618	11444	13738
35-39	4649	5517	6522	7688	9080	10804
40-44	3734	4423	5200	6069	7080	8338
45-49	2972	3513	4109	4746	5454	6325
50-54	2330	2761	3238	3744	4281	4905
55-59	1789	2122	2510	2932	3382	3874
60-64	1327	1574	1862	2191	2552	2950
65-69	930	1103	1307	1541	1811	2120
70-74	593	707	848	1016	1213	1446
75+	515	607	736	905	1117	1377
FEMALES TOTAL	100000	118844	139190	160093	181478	204410
0-4	19791	23489	25893	27936	30120	32775
5-9	15642	18513	21931	23901	25501	27602
10-14	12811	15291	18150	21564	23567	25218
15-19	10557	12574	15040	17890	21300	23329
20-24	8671	10330	12331	14783	17625	21032
25-29	7095	8441	10046	11945	14265	17008
30-34	5782	6869	8133	9569	11248	13393
35-39	4692	5570	6575	7669	8883	10384
40-44	3787	4497	5306	6171	7083	8153
45-49	3042	3613	4273	4989	5739	6562
50-54	2422	2877	3408	3998	4629	5313
55-59	1896	2253	2675	3153	3682	4264
60-64	1440	1713	2041	2422	2855	3345
65-69	1037	1235	1480	1774	2119	2518
70-74	684	817	985	1195	1449	1753
75+	649	762	922	1135	1411	1761
BIRTH RATE		50.0	47.0	44.6	42.6	40.5
DEATH RATE		15.5	15.5	16.6	17.3	16.4
RATE OF NAT. INC.		3.44	3.15	2.80	2.53	2.41
NET MIGRATION RATE		.0	.0	.0	.0	.0
GROWTH RATE		3.44	3.15	2.80	2.53	2.41
TOTAL FERTILITY		7.300	6.800	6.300	5.800	5.300
NRR		2.639	2.446	2.183	1.954	1.821
e(0) - BOTH SEXES		50.37	49.88	47.67	46.09	47.06
e(10) - BOTH SEXES		52.60	52.27	51.16	50.24	50.40
e(20) - BOTH SEXES		44.43	43.88	42.53	41.40	41.37
IMR - BOTH SEXES		106.8	108.2	116.3	116.6	104.7
q(5) - BOTH SEXES		.1759	.1827	.2086	.2270	.2153
DEP. RATIO	103.2	103.0	100.0	94.4	86.9	81.4

Table 8 HIV and AIDS cases, for adults and children, by year: High variant

	HIV incidence (percent)		HIV prevalence (per thousand)		Cumulative HIV cases (thousands)			Current AIDS cases (thousands)			Cumulative AIDS cases (thousands)		
Year	Adult female	Adult male	Adult female	Adult male	Adult female	Adult male	Children	Adult female	Adult male	Children	Adult female	Adult male	Children
1985	17.5	40.6	2.0	4.7	1.0	2.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
1986	37.3	163.3	5.7	20.9	3.0	11.0	0.1	0.1	0.2	0.1	0.1	0.2	0.1
1987	68.3	103.5	12.2	30.5	6.8	16.7	0.4	0.2	0.7	0.2	0.2	0.8	0.2
1988	133.0	129.5	24.9	41.4	14.4	23.9	1.0	0.4	1.0	0.5	0.6	1.4	0.6
1989	179.7	176.4	41.4	56.4	24.9	34.0	2.4	0.9	1.4	1.1	1.2	2.4	1.5
1990	123.4	139.5	50.8	66.3	32.5	42.2	4.6	1.5	2.0	2.0	2.3	3.7	3.1
1991	70.8	85.6	54.4	70.6	36.9	47.3	7.3	1.8	2.5	3.0	3.5	5.3	5.3
1992	49.8	59.2	55.4	71.8	40.0	50.8	10.3	2.1	2.8	3.8	4.7	7.0	7.8
1993	44.1	51.2	55.5	71.9	42.9	53.9	13.3	2.4	3.2	4.1	6.2	8.9	10.5
1994	43.7	49.2	55.5	71.6	45.7	56.8	16.3	2.7	3.6	4.3	7.8	11.0	13.2
1995	44.2	48.7	54.9	70.6	48.6	59.7	19.4	3.1	4.0	4.3	9.7	13.4	15.9
1996	46.2	50.8	54.6	69.9	51.7	62.7	22.4	3.5	4.4	4.3	11.8	16.0	18.6
1997	47.3	52.6	54.2	69.1	54.9	65.9	25.3	3.9	4.8	4.3	14.1	18.7	21.3
1998	48.4	54.7	53.8	68.3	58.3	69.2	28.2	4.2	5.0	4.3	16.5	21.6	24.0
1999	49.2	56.9	53.4	67.5	61.7	72.6	31.1	4.4	5.2	4.2	19.0	24.5	26.7
2000	49.5	58.7	52.6	66.5	65.2	76.1	34.0	4.6	5.3	4.2	21.6	27.4	29.3
2001	50.7	60.9	52.2	66.1	68.8	79.8	36.8	4.7	5.3	4.2	24.2	30.3	31.9
2002	51.1	61.8	52.0	65.7	72.5	83.6	39.5	4.7	5.2	4.1	26.7	33.1	34.5
2003	51.4	62.6	51.8	65.4	76.3	87.4	42.3	4.7	5.2	4.1	29.3	35.9	37.0
2004	51.6	63.3	51.6	65.3	80.2	91.3	45.0	4.7	5.0	4.1	31.8	38.6	39.5
2005	51.4	63.6	51.1	64.7	84.1	95.3	47.7	4.7	5.0	4.1	34.4	41.2	42.0
2006	52.3	65.6	51.1	64.4	88.2	99.5	50.4	4.6	4.9	4.1	36.9	43.8	44.6
2007	52.1	66.0	51.1	64.4	92.3	103.7	53.1	4.6	4.8	4.1	39.4	46.4	47.1
2008	52.4	66.3	50.9	64.3	96.5	107.9	55.8	4.6	4.8	4.1	41.9	49.0	49.6
2009	52.8	66.7	50.5	64.0	100.7	112.3	58.4	4.6	4.7	4.0	44.4	51.5	52.1
2010	53.0	67.3	50.6	64.3	105.1	116.6	61.0	4.6	4.7	4.0	46.9	54.1	54.5

Table 9 Population projection with AIDS: High variant

AGE GROUP	1985	1990	1995	2000	2005	2010
TOTAL M+F	200000	236891	269480	301845	339277	382625
MALES TOTAL	100000	118319	134342	150183	166731	190470
0-4	20102	23629	24247	26232	29324	32044
5-9	15846	18742	21118	21270	23469	26796
10-14	12970	15482	18362	20747	20955	23187
15-19	10679	12716	15208	18074	20462	20709
20-24	8726	10398	12410	14878	17724	20114
25-29	7088	8420	9969	11865	14283	17135
30-34	5750	6797	7921	9275	11101	13542
35-39	4649	5482	6311	7230	8512	10368
40-44	3734	4379	4928	5466	6283	7624
45-49	2972	3468	3815	4052	4482	5371
50-54	2330	2733	3046	3215	3401	3877
55-59	1789	2109	2415	2648	2804	3014
60-64	1327	1561	1783	1999	2209	2391
65-69	930	1093	1246	1394	1581	1795
70-74	593	704	828	948	1079	1252
75+	515	607	733	888	1061	1255
FEMALES TOTAL	100000	118573	135138	151662	170547	192154
0-4	19791	23325	23900	25801	28784	31401
5-9	15642	18495	20867	20957	23065	26273
10-14	12811	15291	18132	20516	20664	22808
15-19	10557	12574	15040	17872	20265	20455
20-24	8671	10330	12331	14783	17608	20009
25-29	7095	8430	9939	11782	14144	16974
30-34	5782	6850	7932	9176	10872	13239
35-39	4692	5551	6363	7175	8268	9968
40-44	3787	4481	5128	5719	6416	7519
45-49	3042	3603	4160	4672	5196	5897
50-54	2422	2870	3332	3791	4253	4781
55-59	1896	2249	2626	3020	3442	3902
60-64	1440	1710	2013	2343	2707	3119
65-69	1037	1234	1470	1740	2042	2386
70-74	684	816	982	1184	1419	1688
75+	649	762	921	1133	1402	1735
BIRTH RATE		50.0	47.5	45.9	44.2	41.7
DEATH RATE		16.2	21.7	23.2	20.9	17.6
RATE OF NAT. INC.		3.39	2.58	2.27	2.34	2.41
NET MIGRATION RATE		.0	.0	.0	.0	.0
GROWTH RATE		3.39	2.58	2.27	2.34	2.40
TOTAL FERTILITY		7.300	6.800	6.300	5.800	5.300
NRR		2.611	2.118	1.878	1.830	1.797
e(0) - BOTH SEXES		49.32	41.39	38.66	41.04	45.28
e(10) - BOTH SEXES		51.74	47.76	45.26	45.75	48.55
e(20) - BOTH SEXES		43.54	39.25	36.51	36.84	39.51
IMR - BOTH SEXES		112.6	147.9	139.9	120.8	105.9
q(5) - BOTH SEXES		.1819	.2723	.2939	.2582	.2217
DEP. RATIO	103.2	103.0	97.2	89.8	84.0	82.2

Table 10 New HIV cases (thousands) by subgroup and year: Low variant

	Adult female subgroups					Adult male subgroups				
	1	2	3	4	5	1	2	3	4	5
1980	0.01	0.00	0.01	0.09	0.03	0.01	0.00	0.00	0.03	0.10
1981	0.01	0.00	0.01	0.08	0.03	0.01	0.00	0.00	0.03	0.09
1982	0.01	0.00	0.01	0.08	0.03	0.01	0.00	0.00	0.03	0.08
1983	0.01	0.00	0.01	0.08	0.03	0.01	0.00	0.00	0.03	0.07
1984	0.01	0.00	0.01	0.08	0.03	0.01	0.00	0.00	0.03	0.07
1985	0.01	0.00	0.01	0.07	0.03	0.01	0.00	0.00	0.03	0.06
1986	0.01	0.00	0.01	0.07	0.03	0.01	0.00	0.00	0.03	0.06
1987	0.01	0.00	0.01	0.07	0.03	0.01	0.00	0.00	0.03	0.05
1988	0.01	0.00	0.01	0.06	0.03	0.01	0.00	0.00	0.03	0.05
1989	0.01	0.00	0.01	0.06	0.03	0.01	0.00	0.00	0.02	0.04
1990	0.01	0.00	0.01	0.06	0.03	0.01	0.00	0.00	0.02	0.04
1991	0.01	0.00	0.01	0.05	0.03	0.01	0.00	0.00	0.02	0.04
1992	0.00	0.00	0.01	0.05	0.02	0.01	0.00	0.00	0.02	0.03
1993	0.00	0.00	0.00	0.05	0.02	0.01	0.00	0.00	0.02	0.03
1994	0.00	0.00	0.00	0.04	0.02	0.01	0.00	0.00	0.02	0.03
1995	0.00	0.00	0.00	0.04	0.02	0.01	0.00	0.00	0.02	0.03
1996	0.00	0.00	0.00	0.04	0.02	0.01	0.00	0.00	0.02	0.02
1997	0.00	0.00	0.00	0.04	0.02	0.01	0.00	0.00	0.02	0.02
1998	0.00	0.00	0.00	0.03	0.02	0.00	0.00	0.00	0.02	0.02
1999	0.00	0.00	0.00	0.03	0.02	0.00	0.00	0.00	0.01	0.02
2000	0.00	0.00	0.00	0.03	0.01	0.00	0.00	0.00	0.01	0.02
2001	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.02
2002	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.02
2003	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2004	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2005	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2006	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2007	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2008	0.00	0.00	0.00	0.02	0.01	0.00	0.00	0.00	0.01	0.01
2009	0.00	0.00	0.00	0.01	0.01	0.00	0.00	0.00	0.01	0.01
2010	0.00	0.00	0.00	0.01	0.01	0.00	0.00	0.00	0.01	0.01

Note: The male subgroups are (1) single; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous with nonprostitutes; and (5) married, promiscuous with prostitutes. The female subgroups are (1) single, nonprostitute; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous; and (5) single, prostitute. with prostitutes.

Table 11 New HIV cases (thousands) by subgroup and year: Medium variant

	Adult female subgroups					Adult male subgroups				
	1	2	3	4	5	1	2	3	4	5
1980	0.00	0.00	0.00	0.02	0.02	0.00	0.00	0.00	0.00	0.04
1981	0.00	0.00	0.00	0.02	0.04	0.00	0.00	0.00	0.01	0.06
1982	0.01	0.00	0.00	0.03	0.09	0.01	0.00	0.00	0.01	0.13
1983	0.01	0.00	0.01	0.05	0.17	0.01	0.00	0.00	0.02	0.26
1984	0.01	0.00	0.01	0.08	0.30	0.02	0.00	0.00	0.03	0.49
1985	0.02	0.00	0.02	0.13	0.47	0.03	0.00	0.00	0.05	0.88
1986	0.04	0.01	0.04	0.23	0.59	0.04	0.00	0.00	0.07	1.38
1987	0.06	0.01	0.07	0.38	0.57	0.07	0.01	0.00	0.12	1.78
1988	0.09	0.01	0.11	0.60	0.44	0.10	0.01	0.01	0.18	1.88
1989	0.14	0.02	0.14	0.89	0.34	0.14	0.01	0.01	0.27	1.70
1990	0.20	0.02	0.17	1.23	0.30	0.20	0.01	0.01	0.38	1.44
1991	0.26	0.03	0.19	1.61	0.29	0.27	0.01	0.01	0.52	1.27
1992	0.33	0.03	0.20	1.97	0.29	0.35	0.02	0.01	0.69	1.16
1993	0.41	0.04	0.20	2.31	0.29	0.44	0.02	0.02	0.87	1.10
1994	0.48	0.04	0.20	2.60	0.30	0.53	0.02	0.02	1.06	1.07
1995	0.54	0.05	0.21	2.81	0.30	0.60	0.03	0.02	1.24	1.06
1996	0.60	0.05	0.21	2.98	0.31	0.67	0.03	0.03	1.41	1.09
1997	0.64	0.06	0.21	3.04	0.31	0.72	0.04	0.03	1.55	1.10
1998	0.67	0.06	0.21	3.02	0.32	0.75	0.04	0.03	1.66	1.12
1999	0.69	0.07	0.22	2.95	0.32	0.77	0.04	0.03	1.72	1.13
2000	0.69	0.07	0.22	2.85	0.32	0.76	0.05	0.04	1.75	1.14
2001	0.69	0.08	0.23	2.79	0.33	0.76	0.05	0.04	1.78	1.16
2002	0.69	0.09	0.23	2.70	0.33	0.75	0.06	0.04	1.78	1.17
2003	0.68	0.09	0.24	2.61	0.34	0.73	0.06	0.04	1.76	1.17
2004	0.67	0.10	0.24	2.53	0.34	0.71	0.06	0.04	1.73	1.17
2005	0.65	0.10	0.25	2.47	0.34	0.68	0.07	0.04	1.70	1.17
2006	0.66	0.11	0.25	2.50	0.36	0.68	0.07	0.05	1.69	1.19
2007	0.66	0.11	0.26	2.49	0.37	0.67	0.07	0.05	1.67	1.21
2008	0.66	0.11	0.26	2.49	0.38	0.66	0.07	0.05	1.66	1.23
2009	0.66	0.11	0.26	2.50	0.39	0.65	0.08	0.05	1.65	1.25
2010	0.66	0.12	0.26	2.52	0.40	0.65	0.08	0.05	1.64	1.27

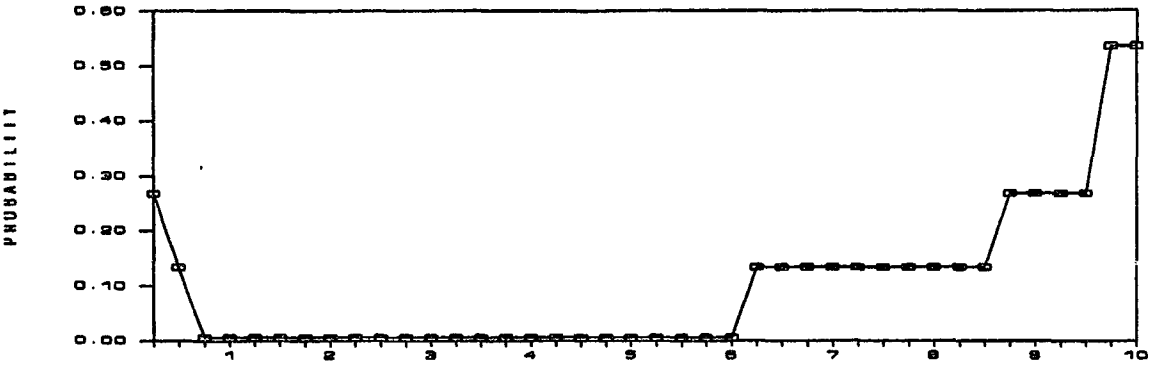
Note: The male subgroups are (1) single; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous with nonprostitutes; and (5) married, promiscuous with prostitutes. The female subgroups are (1) single, nonprostitute; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous; and (5) single, prostitute. with prostitutes.

Table 12 New HIV cases (thousands) by subgroup and year: High variant

	Adult female subgroups					Adult male subgroups				
	1	2	3	4	5	1	2	3	4	5
1980	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1981	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1982	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
1983	0.00	0.00	0.00	0.00	0.01	0.00	0.00	0.00	0.00	0.03
1984	0.00	0.00	0.00	0.01	0.10	0.01	0.00	0.00	0.01	0.29
1985	0.02	0.00	0.01	0.08	0.79	0.05	0.00	0.00	0.03	1.99
1986	0.10	0.01	0.10	0.46	1.32	0.33	0.00	0.00	0.19	8.09
1987	0.53	0.02	0.56	2.30	0.37	0.97	0.01	0.01	0.86	3.79
1988	1.24	0.03	0.76	5.24	0.30	2.34	0.02	0.04	3.31	1.54
1989	1.94	0.04	0.76	7.53	0.28	2.60	0.03	0.09	6.12	1.24
1990	1.47	0.06	0.74	5.02	0.25	1.25	0.04	0.17	5.65	1.07
1991	0.80	0.08	0.72	2.58	0.24	0.62	0.05	0.23	3.18	1.01
1992	0.49	0.10	0.70	1.64	0.23	0.43	0.07	0.23	1.84	0.97
1993	0.39	0.11	0.68	1.42	0.23	0.42	0.08	0.21	1.37	0.98
1994	0.37	0.12	0.65	1.46	0.24	0.47	0.09	0.20	1.16	1.03
1995	0.38	0.13	0.64	1.53	0.24	0.51	0.09	0.19	1.06	1.07
1996	0.41	0.14	0.64	1.65	0.26	0.54	0.10	0.18	1.09	1.13
1997	0.44	0.14	0.63	1.74	0.26	0.56	0.10	0.17	1.15	1.16
1998	0.46	0.14	0.63	1.81	0.27	0.58	0.11	0.17	1.23	1.19
1999	0.48	0.14	0.62	1.88	0.28	0.60	0.11	0.17	1.31	1.23
2000	0.50	0.15	0.63	1.94	0.29	0.61	0.11	0.16	1.38	1.26
2001	0.53	0.15	0.64	2.03	0.30	0.64	0.11	0.17	1.46	1.31
2002	0.54	0.15	0.65	2.08	0.30	0.65	0.11	0.17	1.50	1.33
2003	0.55	0.15	0.65	2.13	0.31	0.66	0.12	0.17	1.55	1.35
2004	0.57	0.15	0.66	2.17	0.31	0.67	0.12	0.17	1.59	1.38
2005	0.58	0.15	0.67	2.22	0.32	0.69	0.12	0.17	1.62	1.40
2006	0.60	0.15	0.69	2.30	0.33	0.71	0.12	0.18	1.68	1.46
2007	0.61	0.15	0.70	2.33	0.33	0.72	0.12	0.18	1.72	1.47
2008	0.62	0.15	0.71	2.37	0.34	0.73	0.12	0.18	1.74	1.49
2009	0.63	0.16	0.72	2.41	0.35	0.74	0.12	0.18	1.76	1.51
2010	0.64	0.16	0.73	2.45	0.35	0.75	0.12	0.19	1.79	1.53

Note: The male subgroups are (1) single; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous with nonprostitutes; and (5) married, promiscuous with prostitutes. The female subgroups are (1) single, nonprostitute; (2) married, monogamous with monogamous spouse; (3) married, monogamous with promiscuous spouse; (4) married, promiscuous; and (5) single, prostitute. with prostitutes.

Figure 5 Assumed probability of transmission through sexual contact, by years since initial infection, for high variant



Alternative projections

These results incorporate the effects of a few parameters unspecified in the standard set. Two are of particular importance: infective phases and the proportion moving among subgroups. Separate runs were made incorporating different values for these parameters, using the high and the medium variants. The low variant is of little interest because infection rates are so low as to show little change under alternative conditions.

One set of runs allowed infectiousness to vary with duration since initial infection, at the same time keeping the average probability of transmission constant. Figure 5 shows how the transmission probability was allowed to vary for the high variant, where an average of .1 was maintained. Between the lowest and the highest probabilities, a ratio of roughly 1 to 100--corresponding to the ratio between low-variant and high-variant parameters--was introduced.

This pattern delays the spread of infection and leads to lower levels of seroprevalence, as Figure 6 shows. Similar differences exist for other output parameters. This is a consequence

Figure 6 Percent of females seropositive when infective phases are defined for high and medium variants

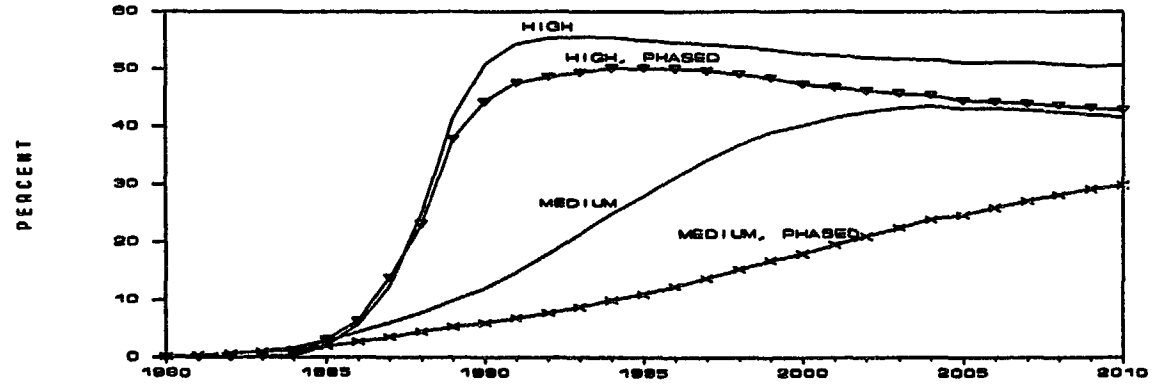
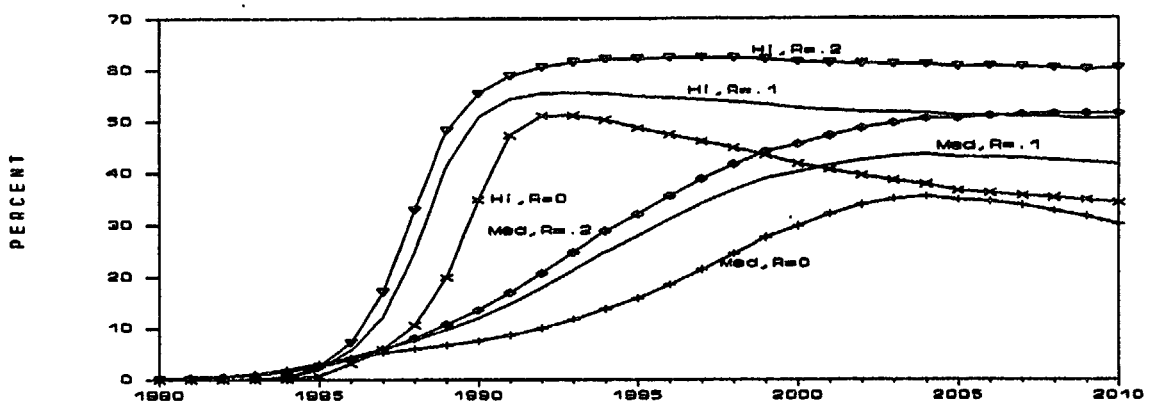


Figure 7 Percent of females seropositive with 0, 10, or 20 percent random subgroup movement, for high and medium variants



of the assumption that high infectivity at the beginning of the incubation period is more than outweighed by a longer high-infectivity phase toward the end.

In the base high and medium variants, it was assumed that 10 percent of the members of all subgroups change membership every year, with their destinations being chosen randomly. Alternative runs were made reducing this random movement to zero and raising it to 20 percent. Figure 7 shows that seroprevalence varies substantially under these alternative conditions.

SIMPLE IMPLICATIONS OF THE MODEL

Simple relationships can be extracted from the model by running it several times with different parameters. For instance, consider the relationship between changes in condom use and HIV infection. The medium variant above assumes that condom use is 10 percent throughout the projection period. From this base, if nonuse of condoms were to be reduced 1 percent annually,

Figure 8 Adult seroprevalence in 2000 and 2010, by decline in condom nonuse

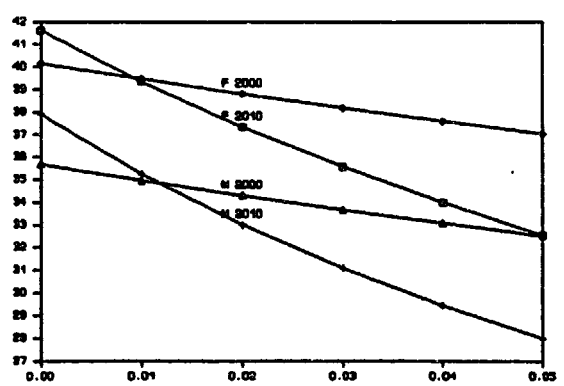
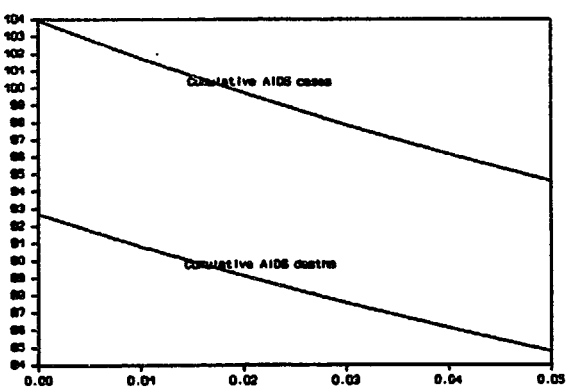


Figure 9 Cumulative AIDS cases and deaths (1000s) by 2010, by decline in condom nonuse



condom use would reach 18 percent in a decade and 26 percent in two decades. On the other hand, if nonuse of condoms were to be reduced as much as 5 percent annually, condom use would reach 43 percent in a decade and 66 percent in two decades. Figures 8 and 9 illustrate the effects of such increases in condom use on HIV seroprevalence and on cumulative AIDS cases. It is assumed that condom use starts to increase in 1990 but that fertility, already on a declining trend, is not further depressed. After a decade, the reductions in the effects of HIV appear relatively small, but after two decades they are quite substantial. An acceleration in the reduction in condom nonuse of 1 percentage point (over the range utilized, and given the other parameters chosen) leads to a 1.5 to 2 percentage point reduction in seroprevalence after 20 years, as well as to a substantial reduction, in the thousands, in cumulative AIDS cases and deaths. Other relationships of this sort can easily be derived with standard parameters.

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